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ACQUIRED CONTRACTURES OF THE HAND*

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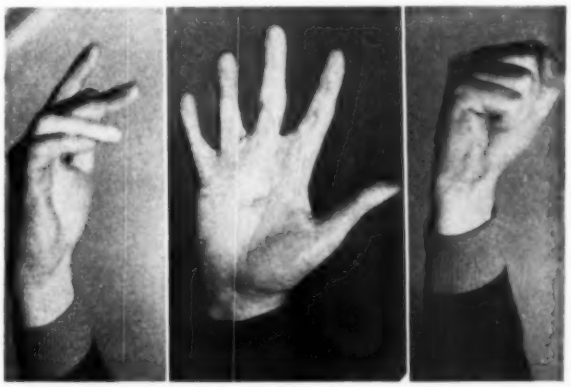
ACQUIRED contractures of the hand, as distinguished from congenital contractures, may be due to a variety of causes, but by far the most frequent, in our experience, are infection and injury. Contractures due to infection and injury may be divided into several groups, corresponding to the anatomical part primarily and chiefly involved. They may be due essentially to a loss of covering tissue; they may be due to fixation of the tendons in scar tissue, or to contraction, division, or actual loss of the tendons; they may be due to perineural fibrosis, or to division of the peripheral nerves in the wrist or hand; they may be due to periarticular fibrosis or to fibrous or bony ankylosis; finally they may be due to an involvement of several or of all the anatomical structures which make up the intricate mechanism we know as the hand.

CONTRACTURES DUE TO A LOSS OF SKIN AND SUBCUTANEOUS TISSUE

Such contractures commonly follow burns and crushing injuries. The essential factor in the treatment is the replacement of firm unyielding scar tissue by normal tissue from some other part of the body.

If the contracture involves the palmar surface of the hand it is usually possible, after complete excision of scar tissue, to cover the raw surface with a free full thickness graft, for as a rule, even after complete excision of contracting scar tissue one still finds a thin layer of tissue covering the sheaths of the flexor tendons, the digital nerves and blood vessels. In such a case after all the scar tissue is excised, the fingers are brought into extension, a pattern of the raw surface is made, and a free full thickness graft of the exact size of the pattern is taken from some other part of the body and accurately sutured over the defect.*



A B C

FIG. 1. Free full thickness graft.

(a) Position of fingers before operation showing marked limitation of extension.

(b) Appearance of hand three months after excision of contracted scar tissue and application of free full thickness graft. Although line of union of graft and skin can be clearly seen grafted skin is soft and supple, and matches well remainder of palmar surface.

(c) Restoration of flexion, as well as extension, is complete.

* We have also found the use of the free full thickness graft of value, as has Blair,[2] in the treatment of webbed fingers. When the fingers are completely and deeply separated a single pattern is made of the entire raw surface and a single free full thickness graft laid over the entire raw surface.

* From the Department of Surgery, Northwestern University Medical School. Read before the Section of Orthopedic Surgery of the New York Academy of Medicine, December 20, 1929.

FIG. 2. Pedunculated flap for burn contracture of dorsum of hand and fingers.

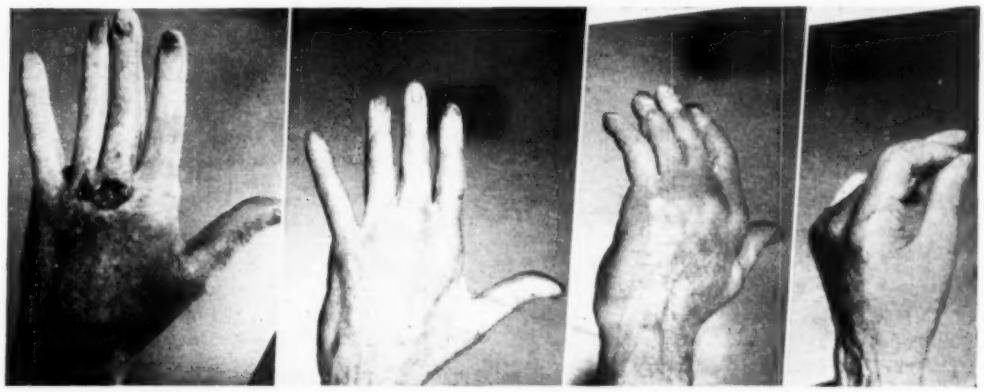
(a, b) Before operation.

(c) Hand underneath flap raised from anterior surface of thigh.

(d) Surface from which flap was raised three weeks after removal of flap, and two weeks after application of Thiersch grafts to raw surface.

(e, f) Appearance of flap just after removal of sutures. (In this case there is complete bony ankylosis of metacarpophalangeal and proximal interphalangeal joints, which is still to be corrected.)

The technic of securing and applying a full thickness graft has been fully described, by Davis,[1] Blair,[2] by ourselves[3] and by many others. An illustration of the results of the application of such a graft is shown in Figure 1.



A B C D

Fig. 3. Pedunculated flap for contracture and ulceration due to *x*-ray burns.

(a) Before operation.

(b, c, d) Four months after excision of scarred and devitalized skin, and application of pedunculated flap from right thigh.

If the contracture involves the interdigital space between the thumb and index finger, or the dorsum of the hand, a pedunculated graft is generally used, for on the dorsum of the hand even a superficial injury usually causes a destruction of all the tissues covering the tendons, and too often the tendons themselves.

Two methods may be used. If the defect to be covered is not too large the tubed flap is very satisfactory. It has the advantage of exposing a minimum area of raw surface to infection from the adjacent skin. If the defect is large and involves the dorsal surface of the fingers the part to be covered is placed under a pocket flap on the abdomen or thigh.*

In placing the hand in such a pocket, a method which has been used and described by many surgeons, and which we have described recently in detail,[3] one should remember the importance of abducting the fingers as widely as possible from one another so that there may be sufficient tissue available for reconstructing the web of the fingers when the flap is sutured to the hand.

The results of the application of such a flap to the dorsum of the hand is shown in Figures 2 and 3.

CONTRACTURES DUE TO INJURY OF THE TENDONS

Injury of the tendons is one of the most common causes of contracture and at the same time one of the most difficult to treat with assurance of success.

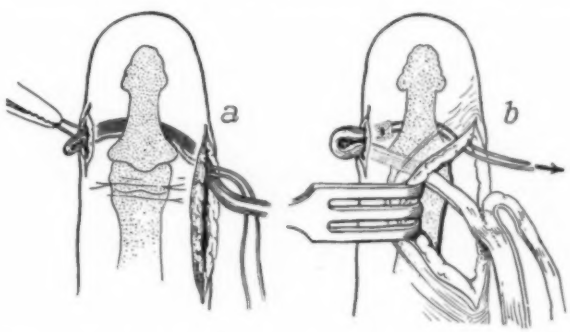


Fig. 4. Method of attaching tendon graft to the distal phalanx of finger.

Several types of tendon injury are of frequent occurrence in surgical practice. The simplest is that in which as a result of a crushing injury the tendons are contused and one or more of the metacarpal bones or phalanges are fractured. Usually there is no open wound. The hand swells

* Blair[4] has emphasized the fact that the skin of the anterior surface of the thigh is thin and soft, and more nearly resembles the normal covering of the dorsum of the hand than the thicker skin of the abdominal wall.

rapidly because of subcutaneous hemorrhage and exudation. Because some of the bones are fractured the hand is immobilized,—too often for a considerable period of time. When attempts at active movement are begun one usually finds the tendons more or less completely fixed by fibrous tissue and flexion of the fingers definitely limited.

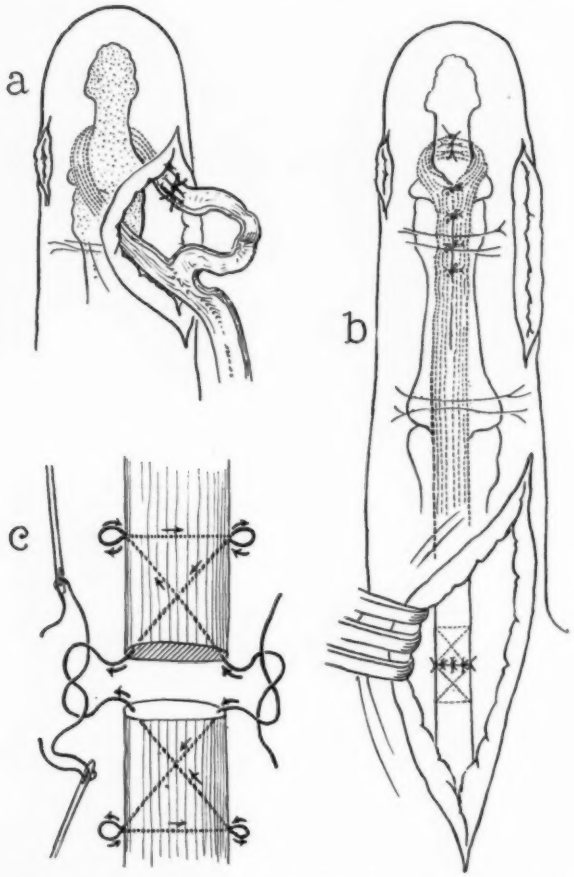


Fig. 5. Method of substituting two tendons from foot for flexor tendons of finger.

(a) Yoke method of attaching tendons to distal phalanx.

(b) Incisions and completed suture. (For clarity finger is shown extended; actually tendon is sutured under some tension and finger is flexed at all joints so that sum of angles of flexion at three joints amounts to approximately 90°.)

(c) Method of attaching tendon graft to flexor profundus digiti.

If there is an open wound with division of one or more of the tendons, usually a tendon suture is performed and, in a certain percentage of cases, wound infection develops. When the wound finally heals active movement is attempted, and again one finds that the affected tendons are bound down by scar tissue and the power of movement is partially or completely lost.

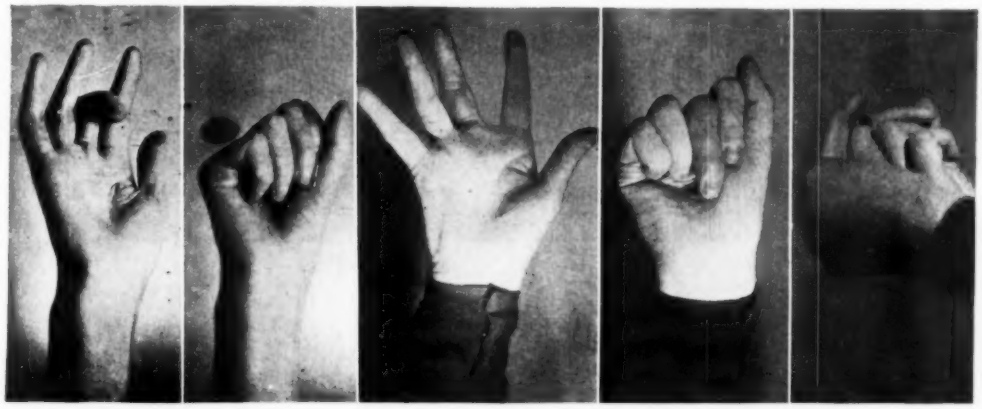
If there is a serious infection of the tendon and tendon sheath the tendon frequently becomes necrotic, a part of it sloughs away and the wound heals only when all of the necrotic tendon is removed.

In the first type of case the best results are secured by instituting passive movements within forty-eight hours after the injury. If the hand and fingers are immobilized for several weeks the tendons become fixed, particularly within the tendon sheaths, and a certain amount of function is lost. Active, persistent and well directed physical therapy, in our experience, constitutes the most satisfactory form of treatment. To free the flexor tendons in the hand and fingers from the surrounding scar tissue by dissection with the hope of securing free movement has not in our hands given satisfactory results.

In the second type of case in which the tendon has been sutured and wound infection has taken place we agree with Bunnell[5] that careful and complete excision of the old tendon, and its replacement by a normal uninjured tendon from the foot give the most satisfactory result. In the third type of case, in which the tendon has been destroyed, replacement of the lost tendon by a normal tendon is the only treatment possible.

In substituting a tendon from the foot for flexor tendons in the fingers one of our problems has been to find a satisfactory method of attaching the new tendon to the distal phalanx. The method which we feel has given us the best results is illustrated in Figures 4 and 5. As a substitute for the flexor tendons of a finger, two tendons with their covering of gliding areolar tissue are taken from the dorsum of the foot. The free end of one tendon is then passed subcutaneously behind the distal phalanx of the finger in question,

and sutured to the free end of the other tendon. By gentle traction the suture line is brought over the middle of the



A B C D E

Fig. 6. Tendon transplantation, following complete loss of flexor tendons in middle finger after suppurative tenosynovitis.

(a) Limit of extension before operation.
(b) Affected finger can be flexed because of action of intact lumbrical muscle.
(c) Extension possible after tendon transplantation. (In this case after excision ofs car tissue remaining at site of tendon and its sheath finger could be extended with little difficulty, but there was a skin defect, roughly triangular in shape and half the size of a postage stamp, over proximal interphalangeal joint. After tendon transplantation this raw surface was covered with a free full thickness graft, as it was impossible to approximate skin edges with finger in extension.)
(d) Flexion possible at all joints after tendon transplantation.
(e) Flexion possible at distal interphalangeal joint.

distal phalanx, and lies well covered by the soft tissues on the dorsum of the distal phalanx. The collar or yoke thus formed about the distal phalanx gives an excellent distal attachment or insertion for the transplanted tendon. Figure 6 shows the results of operation in a case in which the tendons of the foot were substituted for the flexor tendons of the finger according to the method described.

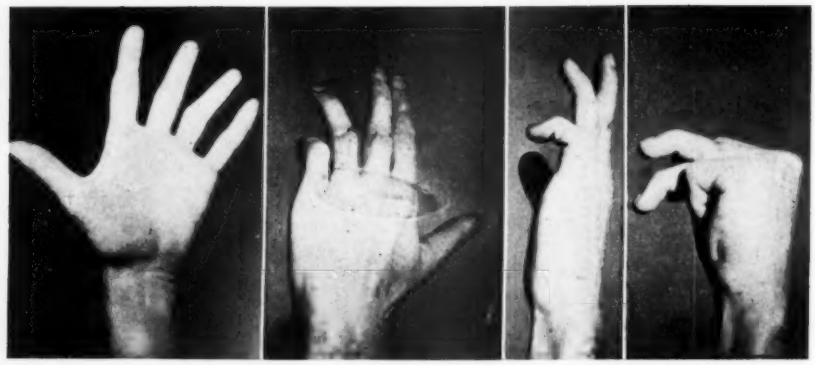
CONTRACTURES DUE TO PERIPHERAL NERVE INJURY

The treatment of contractures due to peripheral nerve injury is too well known to require more than a brief mention. The only logical method of treating nerve division, we believe, is by excision of the

A B C D E

Fig. 7. Division of collateral ligaments of metacarpophalangeal joint for limitation of flexion of fifth finger. (This patient's little finger had been fixed in extension for several weeks after a fracture of metacarpal bone.)

(a, b) Before operation.
(c, d, e) After operation.
Note distance between tip of little finger and palm in (a), and improvement in (c), due almost entirely to increase of flexion at metacarpophalangeal joint.



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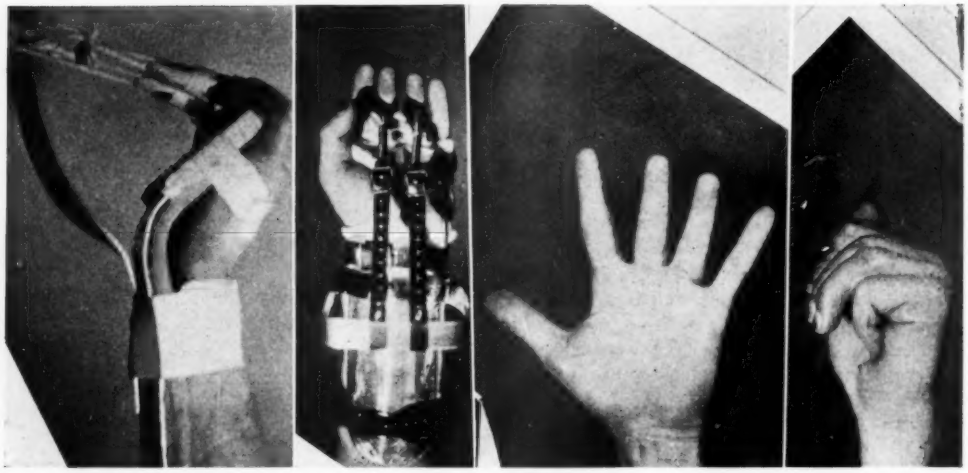

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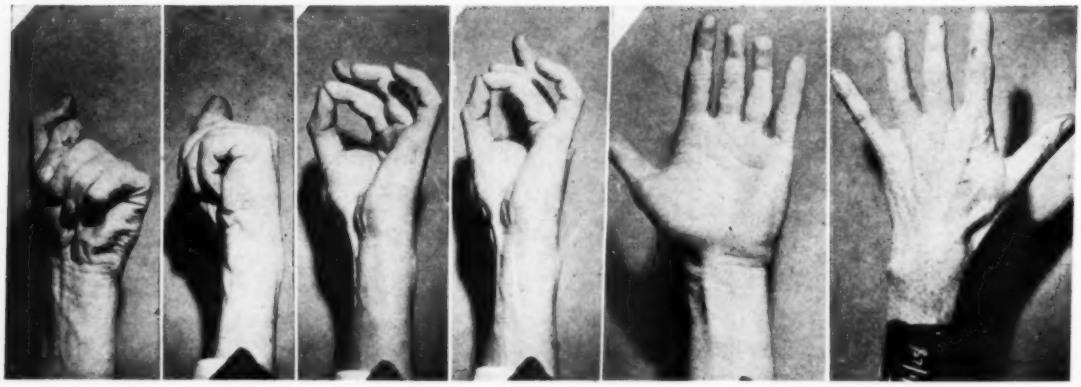

I J K L M N

FIG. 8. Arthroplasty for bony ankylosis of proximal interphalangeal joints of middle and ring fingers.

(This patient, a violinist and teacher, had sustained a saw-cut across dorsum of fingers in a manual training laboratory.)

A, B, C, D. Before operation.

E, F. Method of splinting after operation. G, H. Result four weeks after operation.

I, J, K, L, M, N. Result six months after operation.

neuromata, and careful end to end suture of the divided nerve by uniting the nerve sheaths of the proximal and distal segments with fine silk sutures.

CONTRACTURES DUE TO PERIARTICULAR FIBROSIS AND TO ANKYLOSIS

Contractures due to periarticular fibrosis and to fibrosis and bony ankylosis are common and are frequently neglected. Two types are worthy of special mention. The fixation in extension that occurs, particularly at the metacarpophalangeal joints, as a result of shortening of the collateral ligaments, while the fingers are kept immobilized in an extended position has been described by Shaw[6] and later by Glissan.[7] To the best of our knowledge Shaw was the first to call attention to the fact that the shortened collateral ligaments constitute the most important factor in fixing the fingers in the extended position and that subperiosteal separation of the origin of the ligaments from their proximal attachment permits relaxation and flexion of the stiffened joints.

If bony ankylosis is present arthroplasty is the treatment of choice. Nowhere, in our observation, does arthroplasty give more satisfactory results than at the wrist joint and at the small joints of the hands. At the wrist joint satisfactory results are obtained only if sufficient bone is removed. In our earlier cases we failed to secure a complete range of movement because we removed a layer of bone approximately a half inch in thickness. Of late years we have removed almost twice as much, and with definitely improved results.

Figure 7 shows the improvement obtained in flexion of the little finger by detaching the collateral ligaments from the head of the fifth metacarpal bone, and Figure 8 the result secured by arthroplasty at ankylosed interphalangeal joints.

SUMMARY

Contractures of the hand due to injury and infection may be divided into several groups, in accordance with the anatomical part chiefly involved. Whether skin and subcutaneous tissue, tendons, nerves or joints are chiefly involved definite improvement can frequently be secured by well directed and carefully executed operative procedures. Of these the most important are transplantation of skin by the use of the free full thickness graft and pedunculated flap, tendon transplantation, division of the collateral ligaments of the small joints of the hand and arthroplasty.

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DISCUSSION

Dr. Hugh Auchincloss: Reconstruction work on the hand is difficult enough, but reconstruction of the fingers is far more difficult.

I am sure that we all agree with Dr. Koch that the use of pedunculated grafts and whole thickness grafts is most desirable. If care is taken to remove the fat from beneath whole thickness grafts, nearly all of them will take, provided gentle pressure, freedom from hemorrhage and lack of tension obtain.

It might be wise to distinguish between webbed fingers and fused fingers. Free use of rather thick Thiersch grafts is most desirable if there be any tension, and grafts are excellent for making webs at the base though triangular flaps slung from the dorsum also serve the purpose very well.

Where there has been infection, it is frequently better to use Thiersch grafts to secure immediate healing of defects with the idea of subsequently excising them if necessary and replacing them by whole thickness grafts weeks or months later, when all danger of infection has passed.

To secure motion in a finger where the joints have been preserved but the tendons have sloughed or become adherent, is a fascinating problem and one that so far has not yet been definitely agreed upon by all operators. I must confess that I do not know which of the several different methods is the simplest and best. In my experience the dissection free of an adherent tendon has not been successful. Some form of transplantation must be resorted to. Transplantation of one of the extensor tendons from the foot with its sheath as advocated by Dr. Bunnell, I have done successfully and believe it to be a very good method, but it is technically a difficult procedure. The last few years I have been using transplantations of a portion of the palmaris longus tendon and transplantations of the tensor fascia femoris from the thigh, in both instances preserving the delicate paratendinous tissues. In making such transplantations, there is always the difficulty when sutures are used that the silk sutures will tear out if motion be resorted to early. For this reason I have used very fine artery clamps that have been sharpened to a point so that the sides have a cutting edge. These little instruments permit one to weave tendinous or fascial strips of small size into the periosteal attachments of divided tendons and into the tendons themselves with considerable ease and make what is often a very difficult technical operation much simpler. One of the great difficulties is to provide phalangeal annular ligaments where they have been destroyed so as to prevent tendon prolapse. If the flexor sublimis tendon has its dividing slips along the side available, it is possible to use these. If not, it may be necessary to transplant fascia.

In doing this delicate type of surgery, it is of great advantage sometimes to use magnifying lenses. I have used large magnifying glasses such as reading lenses, but more recently have constructed with a metal frame, so that they can be sterilized, a pair of glasses provided with two magnifying prisms attached by means of an extension to the frame called a Loupe by the occulists. They greatly aid in the recognition of finer structures.

Transplantation of fatty and areolar tissue, especially from along the outer part of the thigh is very useful, particularly about the tendons in the hand.

With reference to arthoplasty of the phalanges, the interposition of fascia, provided enough bone is removed and the cartilage has been entirely lost from the two bones involved, may be of use. Nothing, however, is better than cartilage itself and where one bone involved retains its cartilage, no fascia is necessary. In a recent case, where fibrous ankylosis had taken place between the two proximal phalanges, a portion of bone was excised, the distal end of the proximal phalanx was rounded off and a concavity was bored with a Hudson drill in a costal cartilage in the center of a button that had been cut by means of a trephine. When the button was removed it was possible to round off the convex surface so as to make it resemble the shape of the head of the bone and this little cartilaginous cap fitted nicely over the rounded shaft of the proximal phalanx. The operation was done through a longitudinal incision in the middle of the extensor tendon and motion has been obtained, but there has been some lateral deviation due to the absence of the collateral ligaments. If this be tried again, I shall arrange to leave a little of the sides of the cartilaginous cap cut away so that scar tissue attachments between the side of the bones can take place and serve as lateral ligaments.

Dr. Henry W. Cave: For many years while working in the Dispensary at Roosevelt Hospital I have been forcefully struck by the great number of deformities that follow acute infections of the hand. These deformities seem almost beyond the stage of any remedy. I should like to take this opportunity to emphasize the importance of the removal of both rows of the carpal joints in those patients that have complete ankylosis. They are the most difficult cases that we have to deal with in late results following infections of the hand.

Physiotherapy has given us excellent results.

How long does Dr. Koch carry out physiotherapy and keep up elastic tension before resorting to operative methods?

Recently I had under my care two trained nurses with ankylosis of the metacarpophalangeal joints. Fortunately, I got them early enough, so that with well directed massage and physiotherapy they both are getting good results, and I do not believe that an operative measure will be necessary. One of them had marked ankylosis of the middle, ring and little fingers. I loosely applied elastic tension, and was able to draw out these three fingers into practically a straight line with complete flexion and extension.

Dr. A. R. MacAusland: The courage that

is necessary to follow through this sort of work and get results cannot be appreciated by anyone who has not been confronted by a number of such cases. The contracture deformities and loss of function due to damage of the deeper structures, we, as orthopedic men, are familiar with. I hope to seem more of the tendon grafts done and hope to see more success, but my work in that particular field has been discouraging.

In regard to nerve suture, there is little question but that early nerve suture is the only way to get a highly satisfactory result; the later it is done, the less satisfactory.

In grafting the nerve, little has been accomplished. I have seen and followed through a great many cases, and have not seen a single one the slightest encouraging. The oldest case I had a chance to follow up was a soldier in the Boer War, who had a graft with a rabbit's nerve and that did not show anything at all. One would not expect it to do so. I have had the pleasure, however, of dissecting the section of that particular nerve and having it examined, and excepting for the ordinary scar tissue there was nothing extraordinary in the nerve bulb at the end of the ulnar nerve. So I do not expect that nerve grafts are going to succeed unless some one develops an extraordinary technic. In joints, however, we are improving all the time. Long ago there were very few who attempted arthroplasty, but now many are doing them. The larger joints are the easier ones. The value of early mobilization was emphasized by Dr. Koch, and while I agree that the large resection of bone assures a little more certainty of a mobile wrist, I do not think it is entirely necessary for success. Resection of a joint does not constitute a true arthroplasty, although such an operation may be very successful in mobilizing the joint. We believe that motion and stability can both be obtained by proper operative technic.

Dr. Leo Mayer: When I was talking to Dr. Carrel a few years ago, he told me that he considered reparative work on the fingers the most difficult in all surgery; transplanting of the kidney was a little thing compared with repairing a damaged finger.

I wish to talk about tendons and nerves; first, free nerve transplantation. I have done 6 cases, 4 for the musculospiral nerve in war injuries, where a large portion of the nerve was shot away and it was impossible to get end to end apposition. In 2, perfect restoration of function resulted; in these, I used a cable graft, about 4 in. long, employing the external saphenous nerve in 1 case and the radial nerve in the other. In 2 cases I did the same operation for a long gap in the ulnar nerve and in 1 there was perfect restoration. In other words, of 6 cases 3 had return of normal function.

Dr. Mather Cleveland: The surgery of the upper extremity is the surgery of the hand, to preserve or restore to the individual the prehensile and tactile fingers. Many of the cases that Dr. Koch presented emphasized the fact that hand infections are still treated badly in the beginning.

It has been our endeavor to teach our medical students intensively as Dr. Auchincloss taught us the importance of hand infections, recognizing them early and draining them accurately and adequately in the beginning. Attention to this would do away with the necessity for much of this reconstructive surgical work.

It is important to appreciate the fine details of the anatomy of the hand in order to do this type of surgery properly. One of the most crippling injuries we see is that due to division of the deep branches of the ulnar nerve, with paralysis of the lumbricales and interossei muscles.

In a case of Dr. Krida a large part of the disability is due to the intrinsic muscle paralysis. It is very disconcerting to adequately drain an infected ulnar bursa and in doing so divide the deep branch of the ulnar nerve. This nerve and the branch of the median to the thenar muscles are unfortunately injured in palmar incisions placed without due regard to the anatomy of the region.

I am much interested in webbed fingers and in the method of repair used by Dr. Koch. In the treatment of this condition the important factor is to get good skin at the bottom of the web, and you get that in congenitally webbed fingers by turning down from the dorsum a triangular flap of skin from the web and suturing it into the defect. On the sides may be placed full thickness or Thiersch grafts; but unless you get the bottom of the cleft covered with skin, your web operation will not succeed. I should like to ask how Dr. Koch prepares the skin. I am sorry he did not mention Dupuytren's contraction.

Dr. J. H. Garlock: In the presentation of Dr. Koch's cases of scar contractions of the palm, it was to be noted that the majority of them were corrected by the application of free

full thickness skin grafts following excision of the scar. It has been my experience that the majority of these cases need more tissue than is obtained by free full thickness skin grafts in this situation. Only in an occasional case have I found it feasible to make use of this valuable type of skin grafting. In the majority of instances, I have found it necessary to add subcutaneous tissues as well as skin, and have accomplished this by the use of pedunculated skin flaps in the form of the tubed variety. This method seems so far the most ideal inasmuch as the possibility of contamination and infection is reduced to the minimum. Following the application of such pedicled flaps, it is extremely important to apply an immobilizing spica made of plaster. The arm must be held in position so that the slightest degree of movement is avoided. This can only be done by the use of a plaster cast. I have seen disastrous results follow when this precaution is not taken.

As regards free tendon transplants, the gliding mechanism originally described by Mayer and Biesalski must be taken into consideration. It seems unnecessary and hardly feasible to graft a tendon with its surrounding sheath. A tendon with its surrounding peritenon seems to be the best available tissue for this purpose. Following the insertion of a free tendon graft in the finger proper, it is important to make sure of the presence of vaginal ligaments in order to maintain the finger's mechanical efficiency. Numerous methods for the duplication of these vaginal ligaments have been tried. So far, I have found the most satisfactory ones to be a free tendon graft placed sling-like around the new flexor tendon and the phalanx so as to encircle both structures. Three of these are inserted, one in the region of the middle phalanx, a second about the proximal phalanx, and the third about the head of the metacarpal bone.

The subject of nerve grafting is a very difficult one. I believe that probably the most difficult operation in surgery is that of inserting cable grafts to bridge a defect in a nerve. I have done this operation a few times with very questionable results. One point is certain. It seems absolutely necessary to have a scar-free bed in which these grafts may be placed. Otherwise the contraction of the scar will result in the early strangulation of the nerve grafts. Stookey has emphasized this point. In the treatment of stiffened metacarpophalangeal joints, I find that a great deal can be accomplished by continued elastic contraction and that cutting of the accessory ligaments should be done as a last resort.

Finally, I believe that a word of warning is in order. Reconstruction surgery of the hand must not be undertaken too lightly. It is a very difficult branch of surgery, and is one that is filled with many disappointments and unexpected sequelæ. The surgeon must evolve for himself an operative technic which is well-nigh perfect and which is always cognizant of microscopic trauma. I have found the Beebe-Loup magnifying eyeglass of great help in striving for this minimal tissue trauma.

Dr. Krida: I was very much impressed by Dr. Koch's preliminary skin graft, particularly the full thickness free skin graft, in those cases where a subsequent reconstructive operation on the tendon was indicated. I should like to ask how long after such a graft has been applied can one proceed with the incision of the graft and reconstruction of the underlying tendon.

Dr. Koch, *closing:* Dr. Auchinclos mentioned the importance of covering large raw surfaces on the upper extremity as early as possible with Thiersch grafts. Their value has also been emphasized by Lyle, who pointed out the fact that such grafts lessen the amount of cicatricial contraction and remove the danger of reinfection constantly present in the presence of an open wound.

Dr. Auchinclos asked what method we have used to restore function and the power of flexion if the flexor tendon has been infected. After a severe infection the tendon is usually lost. There remains, however, dense scar tissue at the site of the tendon and its sheath. It has been our custom to dissect out the scar tissue before transplanting a tendon graft. We have not tried the method of introducing a tendon blindly through a small tunnel for we fear it would not move freely in such a tunnel, but become adherent to the surrounding scar tissue.

When the scar tissue has been completely excised along the course of the tendon and the graft has been placed in position one must reconstruct one or more annular ligaments to prevent the tendon from being lifted away from its bed when the finger is bent in flexion. We do not know what is the most satisfactory method of forming such ligaments. We have used pedicled strips of fibrous tissue dissected from alongside the tendon and laid transversely

across the tendon and thin strips of tendon completely encircling the bone.

Dr. M. L. Mason in his experimental work at Northwestern University Medical School on the transplantation of tendons has concluded that the transplantation of the tendon sheath or the gliding tissue about the tendon is an important factor in securing a successful result. Bare tendons do not easily become vascularized, and they tend to become firmly adherent to the surrounding tissues.

I was much interested in the idea of transplanting cartilage into a joint, and shall be interested to know the final result. I recall a patient seen recently in whom the entire proximal phalanx of the thumb was removed because of a giant cell tumor. A piece of rib cartilage was substituted for the removed phalanx. One year later the cartilage had almost completely disappeared.

Dr. Cave asked how long one should continue physical therapy before resorting to operation upon stiff metacarpophalangeal joints. It seems to me that it depends upon the rapidity of improvement. I saw a patient a few days ago who had been operated on for a Dupuytren's contraction and was left with stiffened fingers and hand. We sent her to the physical therapy department and did not see her for two weeks. At the end of that time there was so definite an improvement that we concluded that she would probably secure complete restoration of function if she continued with the treatment. If the fingers are held fixed in extension at the metacarpophealangeal joints, however, I would not hesitate to cut the collateral ligaments.

When I spoke of wide resection of the bone in cases of ankylosis of the joint, I was not speaking of all joints, but only of the wrist joint. I have had no experience with resection of the elbow joint and could not express an opinion as to arthroplasty upon the elbow joint.

I was very much interested in the results Dr. Mayer reported in his cases of nerve transplantation. In some experimental work recently performed at Northwestern University it was shown that nerve fibers will grow down into nerve transplants, but that very few fibers will continue through the transplant into the distal segment of the nerve. Their downgrowth is apparently checked by the fibrous tissue which forms at the distal line of suture. That Dr. Mayer was able to secure successful results in 50 per cent of his cases is extremely interesting.

Dr. Cleveland spoke of the importance of correct surgical treatment of infected hands. I agree absolutely that if cases of infection were properly treated many of the surgical procedures discussed here this evening would be unnecessary. We have been trying in Chicago to teach to every class of students that is graduated from the medical school the principles of the surgical treatment of infections of the hand, as they have been set forth by Dr. Kanavel. We feel the importance of this subject in the surgical curriculum needs to be constantly re-emphasized.

Dr. Cleveland is quite right about the treatment of webbed fingers. If one can get normal skin at the bottom of the interdigital space any remaining defect can be covered with a graft. In suggesting the use of a simple free full thickness graft extending upward on the side of one finger and downward on the opposing side of the adjacent finger I had in mind particularly those cases in which the adjacent fingers are held tightly together, and there is no redundant tissue whatever.

In preparing a hand for operation it is washed with soap and water for twenty minutes the evening before operation, and then wrapped in a sterile dressing. Just before the operation the hand is painted with 5 per cent picric acid in 95 per cent alcohol.

The treatment of Dupuytren's contraction is a long story. A paper giving the results of our experience was published in *Surgery, Gynecology* and *Obstetrics* in February, 1929.

Dr. Krida asked how soon after transplanting a flap one could perform the secondary operations necessary. We like to wait three months after healing is complete so that we may be certain there are no bacteria deep in the tissues. If we have primary union of the transplanted flap we sometimes operate at the end of two months, but we prefer to wait for three months if possible.

COMPRESSION FRACTURES OF THE SPINE

LATE RESULTS IN CONSERVATIVE TREATMENT OF UNCOMPLICATED CASES*

FREDERICK CHRISTOPHER, M.D., F.A.C.S.

EVANSTON, ILL.

IN view of the fact that there exists some diversity of opinion in regard to the best method of treatment of uncomplicated compression fractures of the spine, the writer ventures to report the present small series which seems to afford very favorable evidence in behalf of the conservative treatment in contrast to the operative treatment.

Compression or crush fractures of the vertebrae are attracting more interest of late because of their frequent occurrence and because they are often unrecognized. In a series of 400 spinal injuries Sante and McCutcheon[1] found that 40 per cent were compression fractures and, moreover, that of these compression fractures 80 per cent were between the tenth dorsal and the third lumber vertebrae. These writers emphasize the necessity of making both anteroposterior and lateral roentgenograms and call attention to the need of differential *x*-ray diagnosis between tuberculous spondylitis, hypertrophic spondylitis, pyogenic infection of the spine, Charcot's spine, malignant involvement, and pressure destruction of the vertebrae. According to Osgood[2] 50 to 60 per cent of compression fractures involve the twelfth dorsal or the first lumbar vertebrae.

The etiological mechanism in these fractures is hyperflexion of the spine, which occurs particularly in falls. Of late it is more common in women because of automobile accidents. Verneuil[3] reported a case of compression fracture due to muscular action in a patient who made a successful attempt to escape a fall on the ice. The writer is now taking care of an elderly woman with compression fracture of the first lumbar vertebrae which resulted merely from a fall down two marble steps. In a series of 90 spine injuries Stewart[4] found that 42 per cent of the compression fractures had been unrecognized.

Brown and Brown[5] report the case of a compression fracture in a thirteen year old girl, whose back was hurt on a slide and who went on a hike three hours afterward. These writers note that many patients do not consult a physician until three months after the injury.

Mill[6] reports the case of a compression fracture in a girl of seventeen from a very slight trauma while at play and which was due to strain in a moment of relaxation.

In 1895 Kummel[7] reported serious late results in these cases. As Kummel has shown[8] the wedge-shaped deformity of the vertebra may develop in the absence of typical *x*-ray findings at the original examination. Kummel's disease is the painful back with wedge-shaped deformity of the vertebra which develops in cases where there has been an absence of a clinically demonstrable lesion after a fall.

While it is often possible to reduce the deformity fairly well, late *x*-ray studies are apt to show its persistence. Rogers[9] goes so far as to say that "with the most careful treatment and protection possible he has never seen an *x*-ray examination in the later stages that did not show a greater amount of wedging than was shown in the early *x*-ray study." (Compare Figs. 1, 2, and 3.)

Until recently attention was directed chiefly to the avoidance of weight bearing and to fixation of the spine in the treatment of these cases. Stewart[10] recommends three months in bed followed by six months in a brace. Brown and Brown[5] place their patients in extension for two months and follow this with a brace or cast for six to twelve months. Rogers[9] uses a plaster shell

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for six weeks and places the time of average recovery at six months. Eikenbarry[11] keeps his patients in bed on a convex frame for three months and uses a spinal support for nine months more. This writer, who reviewed 131 cases of compression fractures of the vertebrae, states that if a plaster shell is used, it should extend from the head to the knees and that healing requires six to eight months. Scudder[12] advises correction by hyperextension and support for about six months. He believes operation to be unnecessary. Osgood[13] who is a strong advocate of the non-operative treatment advises recumbency in hyperextension for six to eight weeks. The upright position is then gradually assumed and a spine brace is worn for ten to sixteen weeks. In the last five to six weeks Osgood advises physical therapy and says that a full cure may be expected. Operative treatment, he observes, is rarely indicated, and even in the old cases conservative methods are to be tried first.

In recent years more stress has been placed upon the need of reduction in these fractures. In January, 1929, Davis[14] reported 11 cases treated by obtaining the limit of hyperextension and maintaining it. With the patient prone the legs are lifted up by suspension until the pelvis clears the table by several inches. Some traction is exerted in the footward direction. A downward thrust is made upon the gibbus if necessary. A plaster shell is then applied from the head to the knees for six to ten weeks and is followed by a Taylor spine brace.

The results obtained by Dunlop and Parker[15] are so striking that their method will be described verbatim.

The reduction of these fractures is accomplished by forcible hyperextension of the back, while strong traction and counter traction

Fig. 1. J. E. October 31, 1928, date of injury, showing compression fracture of first lumbar vertebra.

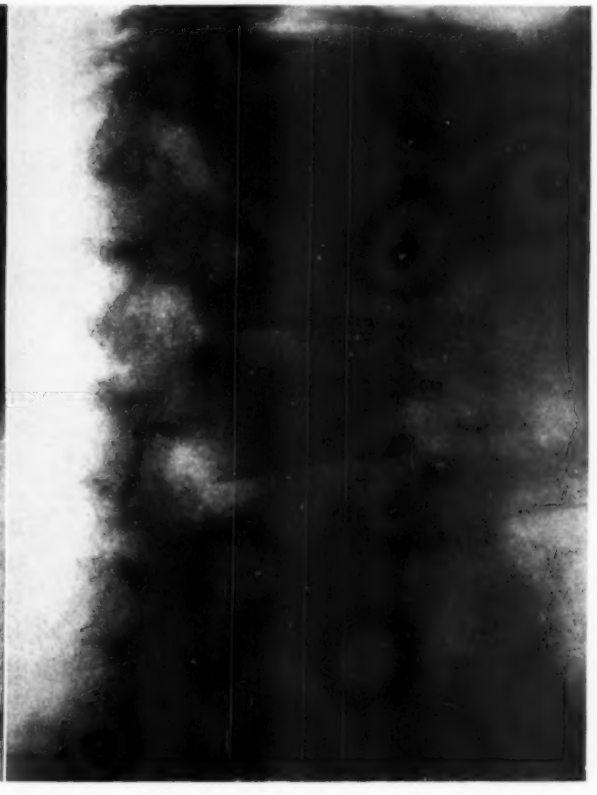

Fig. 2. One week after injury showing reduction of fracture of first lumbar vertebra. Compare with Fig. 1.

were applied to the body. The traction is obtained by two men pulling upward on sheets which are passed diagonally across

Fig. 3. February 25, 1929, nearly four months after injury. Showing poorer position than in Fig. 2.

the chest and under the arm of the opposite side; the counter traction is furnished by two men pulling downward on the ankles. The patient is placed under an anesthetic in the supine position, and a sheet folded to a width of 6 in. is passed beneath the injured segments. The operator and his assistant then toss the patient straight up and catch his weight on the taut sheet while the strong traction is applied to the body. Care is exercised to keep the back in a position of hyperextension. A body cast is then applied with the patient lying on a Goldthwait frame. The procedure may be repeated several times if it seems necessary.

Dunlop and Parker show most convincing *x*-ray studies before and after this treatment. Measurements of the films show the bodies to be actually widened and that this increase in width persists as long as sixteen months after the injuries with the patient back at work.

In the discussion of Dunlop and Parker's paper Dr. Eikenbarry stated that he had seen over 100 of these cases and in not a single case in which a fusion operation was done was the man able to go back to hard work. "The only patients who were able to go back to hard work were those who were treated conservatively." The operative method, however, has its advocates. Speed[16] recommends operation in compression fractures of the lower lumbar vertebrae or "where angulation is increasing."

In 1918 Brackett, Nuxter, and Wilson (quoted by Thomas) reported 27 cases of uncomplicated spinal fractures. Twenty-two of these cases where treated conservatively by recumbency and support and of these 18, or 82 per cent, showed a persistent partial disability. In 9 cases of patients, however who were operated upon, there was complete recovery in every case. Sever (quoted by Thomas) in 1917 reported 27 cases of compression fractures all totally disabled from further heavy duty and Cleary (quoted by Thomas) in 1924 reported 52 cases of compression fracture with an average disability of the unoperated cases of 40 to 50 per cent. In 1928 Thomas[17] reported the end-results of 100 cases of spine injury in a mining community. There were 46 compression fractures in this series with no deaths. Twelve cases were not followed up. In the remaining 34 cases, 15, or 45 per cent, showed no disability; 3, or 9 per cent, showed 5 to 25 per disability; 7, or 20 per cent, showed 25 to 50 per cent disability; 3, or 9 per cent, showed 50 to 75 per cent disability, and 6, or 17 per cent, showed total permanent disability. With partial to total disability in more than half of the cases, Thomas has now adopted the policy of doing a fusion operation, preferably of the Hibbs type, in all compression fractures without cord lesions. He will report his results later.

The present cases are drawn from a series of 21 spinal injuries. Of these 2 were complicated by paralysis, 3 involved the cervical spine, 3 were compression fractures sustained too recently to note

the result, 1 was a compression fracture not seen until one year after the injury, 3 were compression fractures which were not able to be followed, and 9, the series studied, were uncomplicated compression fractures of the dorsal and lumbar vertebrae which were able to be followed up. Table 1 gives the details of these cases.

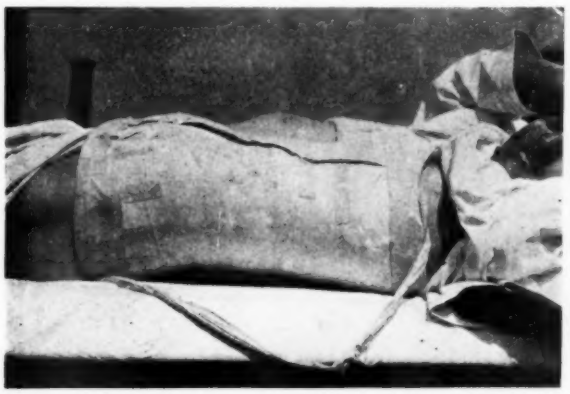

Fig. 4. Plaster body cast applied with patient's face downward and with back in lordosis. Note large window cut out to facilitate breathing, and also "scratcher" bandage passed underneath cast.

Perhaps the most important single factor in the treatment of these cases is proper care of the patient's mental condition. The patient's condition should be described to him truthfully and accurately. He should be told that he has not a broken back in the customary sense of the word, but that he has a lesser injury of one of his vertebrae. He can be told that the prognosis is excellent but that he must follow instructions explicitly.

The usual treatment was to suspend these patients prone in the hammock of an Abbott frame placing the back in hyperextension and to apply a plaster body cast in this position. In some cases, a strong downward thrust was made over the injured vertebra before the cast was applied. After they were placed in bed a large ventral window was cut out of the cast to facilitate respiration and to make greater comfort after eating (Fig. 4). The patients were kept recumbent for at least six weeks and then gradually were permitted to walk about with a well fitted Taylor spine brace. The spine brace was worn from three to nine months and then was gradually removed. In two of the women patients, G. J., and J. O. E., a body cast was not used but the entire treatment was carried out on a Bradford frame in lordosis. The lordosis was brought about by placing under the injury smoothly folded flannel blankets of the proper shape and thickness. More recently the writer has employed upward traction to a Balkan frame to secure hyperextension in the cases of rather fragile elderly women. After the patient is placed on an ordinary Bradford frame an ordinary woman's corset is placed under the site of the injury. The sides of the corset are then trimmed off and attached by numerous adjustable tapes to a 12 to 14 in. wooden spreader bar. The two spreader bars are in turn attached to an overhanging spreader which is suspended from the Balkan frame by a turnbuckle attachment. The turnbuckle is tightened up a little each day during the first week until

Fig. 5. Turnbuckle suspension for compression fracture of spine. Note that spreader bars are attached by adjustable tapes to ribbed corset back. No ribs must be immediately over spinous processes. By means of adjustable tapes and turnbuckle required degree of lordosis is obtained and maintained.

TABLE I

Patient	Sex	Age	Date of Injury	Cause of Injury	Vertebrae Affected	Complications	Treatment	Days in Recumbency	Days in Spine Brace	Interval from Injury to Follow-up	Condition on Follow-up	Result
A. F.	M.	49	7/3/26	Struck by train.	4 L	9 minor fractures of spine, compound fracture l. leg, 15 rib. fractures.	Amputation l. leg, rest in bed, Taylor spine brace.	91	49	3 years, 7 mos.	No pain in back. Not wearing brace. Not doing same work as before (amputation).	G.
G. J.	F.	42	3/18/27	Fall from window.	2 L	Bilateral Colles' fractures.	Rest in lordosis on Bradford frame. Taylor spine brace.	57	180	2 years, 10 mos.	No pain. Doing same work as before. Wears corset.	E.
D. M.	F.	44	5/21/27	Fall from horse.	7 D	None.	Body cast. Taylor spine brace.	39	180	2 years, 8 mos.	No pain. No brace worn. Walks 12–15 miles daily or several sets of tennis.	E.
F. H.	M.	38	5/10/27	Fall from scaffold.	1 L	Fracture middle malleolus left tibia.	Bradford frame, body cast, Taylor spine brace.	63	180	2 years, 8 mos.	No pain except a twinge with sudden twist. Same work as before (bricklayer). No brace but wears belt.	G.
A. P.	M.	26	6/3/27	Fall astride a ladder.	2 L	Rupture of urethra (requiring 2 operations).	Rest in bed, Taylor spine brace.	70	120	2 years, 7 mos.	No pain in back. Same work as before (painter). No brace. "Never felt better in my life."	E.
E. H. M.	M.	46	1/16/28	Fall down icy steps.	1 L	None.	Body cast in lordosis. Taylor spine brace.	35	35	2 years.	No pain in back. Same work as before. No brace. "I am 100 per cent."	E.
C. L.	M.	42	4/24/28	Fall from scaffold.	12 D	Colles' fracture.	Body cast Taylor spine brace.	45	270	1 year, 9 mos.	A "trifle" of pain in the back just above hips when sitting (not at site of fracture). Same work as before. No brace. Spent considerable time in bed for over 1 year.	G.
J. E.	M.	46	10/31/28	Fall from ladder.	1 L	None.	Body cast Taylor spine brace.	49	77	1 year, 3 mos.	Slight pain in back in morning and night. Not doing same work as before injury. No brace or support. Walks as well as before. "Not able to sleep at night."	F.
J. O. E.	F.	61	12/20/28	Fall from stepladder	12 D	None.	Bradford frame with supporting sling. Taylor spine brace.	49	135	1 year, 5 mos.	No pain in back. Doing splendidly. Slight dull ache when tired. No brace. Wears corset.	E.
Average	....	43.7 years	.....	.....	.....	.....	.....	55 days	136 days	27.5 mos.		

the proper hyperextension of the spine is secured (Fig. 5).

After the patient is ambulatory it is important to adjust the Taylor spine brace with brace wrenches so that it accurately conforms to the lordosis curve of the back. A properly made corset, fitted by an expert, will maintain lordosis in a slight woman. It should be supplied with an inner belt.

Analysis of the table shows that the average age of the 9 cases was 43.7 years; the average period of recumbency was fifty-four days; the average time of wearing of the back brace was 136 days; the average time of follow-up report was 27.5 months. The first lumbar vertebrae was involved three times; the second lumbar and twelfth dorsal each twice; and the fourth lumbar and seventh dorsal each one. In 8 cases there is no definite report of pain in the back at the site of the fracture. In the ninth case "no pain except a twinge with a sudden twist" was reported. Seven patients are doing the same work as before the accident. Of the 2 who are not doing the same work, one is prevented from doing so by an amputation of one leg. The seventh patient (C.L.) had apparently the most protracted convalescence saying that for a year he spent considerable time in bed.

In conclusion, the results in the 9 cases treated by conservative measures and followed in an average of 27.5 months, may be summed up as follows:

Excellent	5 (55.5 per cent)	88.8 per cent
Good	3 (33.3 per cent)	
Fair	1 (11.1 per cent)	
Poor	0	

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DEFORMITIES AND OBSTRUCTION OF THE DUODENUM*

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CHARLOTTE, N. C.

THE usual causes of deformities and obstruction of the duodenum are ulcer, gall bladder and periduodenal bands and membranes, diverticula of the duodenum or first loop of jejunum and compression of the duodenum by mesenteric pedicle of the superior mesenteric or median colic arteries.

There is a well defined pathological anatomy and disturbed function produced by these conditions and in the cases of obstruction a clear-cut clinical picture, which *x*-ray studies may confirm with an accuracy of 90 to 95 per cent; even in the cases of ulcer, adhesions from the gall bladder and diverticula, though the clinical picture is not infrequently confusing, persistent *x*-ray studies may finally clear up the diagnosis in almost every case.

A. ULCER OF THE DUODENUM

The typical history of pain or discomfort in the epigastrium or right hypochondrium, when the stomach is empty or emptying its acid contents, with periods of relief and exacerbation of symptoms persisting for months or years and frequently in young healthy looking individuals, is familiar to all. The *x*-ray studies portray varying shadows in the course of development of a callous ulcer from duodenal spasm to well defined filling defect when the ulcer is definitely formed. A progressive obstruction in the second or third portion of the duodenum may hasten ulcer formation and ulcers may form below the duodenal cap.

CASE I. Mr. R. H., aged thirty-five, with typical symptoms of duodenal ulcer who in spite of irregular Sippy and antacid diet, belladonna and alkalies had profuse vomiting of blood, improved and hemorrhage ceased after medical treatment. Filling defect persisted (Fig. 1), with gastroenteroptosis and dilated duodenum. Finney pyloroplasty with resection of ulcer; pathological report of malignant ulcer (discredited). Symptom-free for eighteen months, usual confusing shadow-graft following pyloroplasty (Fig. 2). Sudden recurrence of symptoms and acute perforation; perforation cauterized and closed, gastroenterostomy to relieve tension in duodenum. Symptom-free for fourteen months; recurrence of pain in epigastrium and tenderness, ulcer at gastrojejunal anastomosis (Fig. 13). Free of symptoms for year after short treatment with belladonna, alkalies and antacid diet. Impression: a duodenojejunal anastomosis followed by pyloroplasty and resection of ulcer would have forestalled recurrence of ulcers.

CASE II. Mrs. M., aged sixty-two; pain in epigastrium, occasional vomiting. Two *x*-ray series with barium enemata and visualization of gall bladder, showed only gastroenteroptosis and moderately dilated duodenum. Improvement of short duration on belladonna, alkalies and antacid diet, attacks of pain in epigastrium recurred from time to time over a period of four months' observation; finally passed blood by bowel and vomited blood. *X*-ray study showed dilated and deformed duodenum, (Fig. 4). At operation dilated duodenum, ulcer in second portion of duodenum, relieved of pain by gastroenterostomy for eight months to date. Impression: Same relief of symptoms and likely more enduring comfort by duodenojejunostomy.

CASE III. Mr. M., aged forty-nine, vomited for twenty-four hours voluminously, small bowel movements after repeated enemata, arrived at hospital at night under diagnosis of partial intestinal obstruction. Moderately distended abdomen, scar of former appendectomy, slight tenderness over right hypochondrium, temperature 101°F., pulse 82. Blood pressure 130/70, white blood corpuscles 19,800, few white and red blood cells in urine. Cystoscopy negative, *x*-rays of gastrointestinal tract. Diagnosis: duodenal ulcer perforating (Fig. 5). At operation gall bladder bound

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closely to duodenum, containing mucopus and stones. Liberation and removal of gall bladder. Complete relief.

irritation as there is considerable tendency to spasm of the cap and at times, the peristalsis of the stomach is exceptionally vigorous.

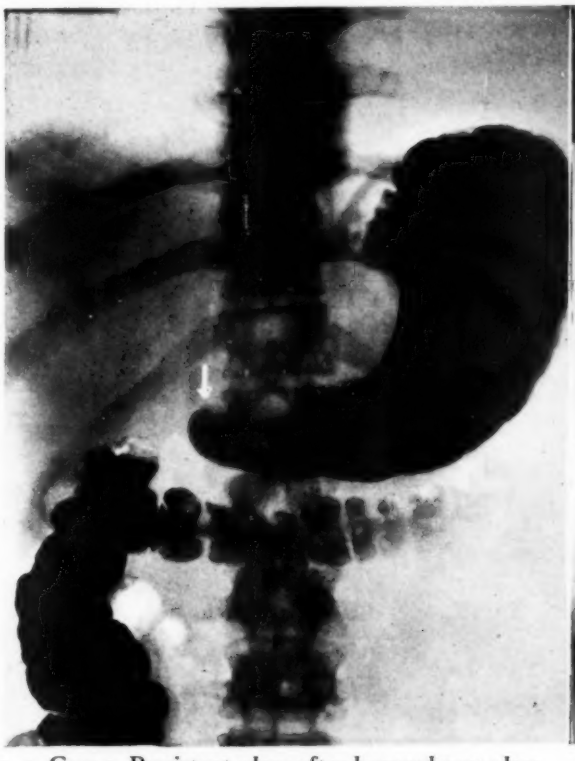

Fig. 1. Case 1. Persistent ulcer after hemorrhage; ulcer, clearly visible and palpable at operation, resected. Finney pyloroplasty.

Fig. 2. Case 1. After Finney pyloroplasty and resection of ulcer, dilatation of duodenum, recurrence of ulcer and perforation. Cauterization and closure of ulcer and gastroenterostomy. Recurrence of ulcer from stasis and tension likely prevented by duodenojeunostomy and resection of ulcer.

Case iv. Mr. A. B., aged thirty-five to forty-five during examinations. At one time symptoms of duodenal ulcer, relieved by belladonna, alkalies and antacid diet; at another time of gall bladder; at another colitis with gas, soft stools or diarrhea with some mucus, always better after thorough purging with salines and on diet with minimum red meats and roughage. Local roentgenologist diagnosed duodenal spasm and spastic colitis; Dr. Fred Baetjer (Baltimore) appendicitis with duodenal spasm; Dr. Pancoast (Philadelphia) adhesions from cholecystitis or duodenal ulcer, poorly drained appendix. The following are the conclusions of Lewis Gregory Cole (New York) after a most elaborate and protracted *x*-ray study:

From a study of these roentgenograms, I believe one is justified in making a negative diagnosis of cancer and ulcer of the stomach and a negative diagnosis of the cap. There is no pyloric stenosis, no functional gastric retention; in fact, the stomach empties more rapidly than usual. There is apparently some

There is a diverticulum of the first part of the descending duodenum. (This diverticulum was never found before in the several *x*-ray studies. Was it acquired or was it always present during these former studies?) This lies to the left of the duodenum and apparently anterior to, and in close relation to the junction of the common bile duct and pancreatic duct.

I find no evidence of calcified gallstones. The gall bladder did not fill with the dye, although this was given orally on two successive days. In our experience this is quite positive evidence that the gall bladder in diseased.

(What relation does the diverticulum bear to failure of the gall bladder to fill, located as it is in close relation to the junction of the common bile duct and pancreatic duct; what would be the difficulties of satisfactory removal at this location and if removed the chances of further obstructing the ducts and finally what are the dangers of such diverticula

beyond a causative agent in the patient's discomfort?)

"I believe one is justified in making a negative diagnosis of a diverticulosis of the lower descending colon and the sigmoid colon. There is no evidence of diverticulitis. The appendix is patent, almost six inches long and apparently contains several small fecoliths. I would consider this a pathological condition of the appendix but I do not believe it is by any means a surgical condition. There is apparently, a congenital veil or band of adhesions in the right hypochrondrium which involves but does not obstruct the hepatic flexure of the colon.

Fig. 3. Case I. After closure of recurrent and perforated duodenal ulcer and gastroenterostomy. Marginal ulcer ½ in. median from arrow point, rapidly emptying stomach and colitis. Gastroenterostomy to be closed, followed by duodeno-jejunostomy.

(This patient with unfilling gall bladder, duodenal diverticulum, pathological appendix and hepatic adhesions remains unoperated on, relieved of the fear of perforating ulcer and ruptured appendix. What surgeon could resist operation on this case?)

B. ADHESIONS FROM GALL BLADDER

Those cases are confused with duodenal ulcer as cited under Case III (Fig. 5), where a gall-bladder visualization was not done either on account of the acuteness of the case or confidence in the diagnosis, when there was an apparent conformity between clinical data and apparent *x*-ray findings. An accurate diagnosis can usually be made when the *x*-ray studies are persistent as in Case IV.

C. PERIDUODENAL BANDS AND MEMBRANES

The supramesocolic bands or membranes are confusing and probably can not be diagnosed clinically or by *x*-ray study with any degree of accuracy and are found at operation under the diagnosis of cholecystitis and duodenal ulcer with adhesions. The inframesocolic bands and adhesions may be projected, as from the symptoms of persistent duodenal obstruction, without relief from position or posture and *x*-ray findings of a dilated and obstructed duodenum.

Case V. Mr. G., aged forty-eight weight 106 lb., loss of 40 lb. in weight, pain in epigastrium, frequent attacks of nausea and voluminous vomiting, pale and weak. *X*-ray study showed dilated duodenum, dilated stomach with eleven hours gastric retention (Fig. 6). At operation bands of adhesions at terminal duodenum and rolling and narrowing of duodenum, dilatation of proximal duodenum. Duodenum liberated and anastomosis made between first loop of jejunum and inframesocolic duodenum. The patient never vomited again, was on a full diet in the course of three weeks and according to his own statement remains perfectly well with a weight of 160 lb., eating what and when he wants. He has remained well for six years. A defect in cap shown in *x*-ray and diagnosed ulcer was not found at operation as a visible and palpable ulcer.

Case VI. Mr. J., aged thirty-eight. This patient, lying on a stretcher, pale, thin, weighing 98 lb., was shown before the local medical society with Mr. G., Case V, standing beside him. The contrast was startling. This case had lost about 50 lb., had severe pain shortly after taking food, frequent attacks of nausea and voluminous vomiting. Had become

a morphine addict. He showed a four plus Wassermann reaction. *X*-ray showed gastroenteroptosis, markedly dilated duodenum in all pictures, gastric retention of twelve hours or more (Fig. 7). He had been receiving antiluetic treatment. At operation there were numerous adhesive bands around the terminal duodenum, this portion was thickened and hard and a definite constriction band was felt in the walls of the intestine, the proximal duodenum markedly dilated. An inframesocolic duodenojejunostomy was done. After operation no more vomiting, there was a gain of 30 lb. in the first month, in spite of an ever positive Wassermann reaction and a continuation of the morphine habit. In course of time the patient developed paresis and finally died with terminal pneumonia but never vomited again.

Fig. 4. Case II. Dilated and deformed duodenum, ulcer in second portion, upper bulging near cap an artefact, after violent hemorrhage. Two former *x*-ray studies negative. Relieved by gastroenterostomy. Stasis and tension in duodenum would have been better provided for by duodenojejunostomy.

Fig. 5. Case III. Artefact produced by gall bladder adhesions. Relieved by liberation of gall bladder from duodenum and cholecystectomy.

Case VII. Miss C. M., aged thirty-two thin, pale, frequent attacks of nausea and voluminous vomiting. *X*-rays showed moderately dilated duodenum and stomach, gastric retention for eleven hours. A gastroenterostomy was done. After operation there was frequent vomiting of large quantities of bile stained fluid, so-called vicious circle. The vomiting was relieved by elevating the foot of the bed. Vomiting frequently occurred during six months and there was no improvement. Repeated *x*-rays showed markedly dilated duodenum and stomach and prolonged gastric retention (Fig. 8). At the second operation the terminal duodenum was found to be surrounded by adhesions and narrowed and the terminal loop of jejunum used for gastroenterostomy carried to the left, made a sharp angle at the duodenojejunal junction. A duodenojejunostomy was done. There was no more vomiting, a rapid gain in weight and marked relief but multiple neurotic complaints, such as irregularity of menstruation, pain in hips, etc. *X*-rays after duodenojejunostomy showed stomach emptying in four hours and rapid passage into cecum (Fig. 9).

Case VIII. Mr. H., aged thirty-four, with pain, frequent nausea and vomiting, loss of appetite and weight, relieved by liberation and unrolling of terminal duodenum from bed of constricting bands, without duodenojejunos-

tomy (Fig. 10). There is a possibility of recurrence of adhesions, though patient remains well for nearly two years.

(Fig. 11). The gastric contents rapidly fills the small intestine and by rapid overloading, vomiting recurs. Impression: At the opera-

Fig. 6. Case v. Dilatation and deformity of fixed obstruction of duodenum by adhesions and rolling of terminal duodenum. Nitch in cap diagnosed ulcer, not visible and palpable at operation. Completely relieved by duodenojejunostomy.

Fig. 7 Case vi. Dilatation and fixed obstruction of terminal duodenum by extrinsic adhesions and intrinsic stricture (4+ Wassermann reaction) completely relieved by duodenojejunostomy.

Case ix. Mrs. McD., aged forty-two. Thin, pale, frequent attacks of voluminous vomiting. History of possible tuberculous peritonitis when a child; formerly had cholecystectomy. *X*-rays showed dilated stomach and duodenum with nine hours' gastric retention, gastroenteroptosis. At operation there were multiple bands at terminal duodenum, marked angulation at duodenojejunal angle, and membrane holding terminal duodenum and proximal jejunum together. At operation, that duodenum and stomach relieved without failure of obstruction, the duodenojejunal angle was liberated from bands and membranes and also from the ligament of Trietz and the anastomosis made between gastric antrum and the duodenojejunal junction. The patient improved, gained weight but soon had an occasional return of attacks of vomiting and some diarrhea. The stomach and duodenum now empty about as fast as the barium meal can be given

tion a duodenojejunostomy would have sufficed. The question is now before us of taking down the gastroenterostomy and making a duodenojejunostomy.

D. DIVERTICULA OF THE DUODENUM AND JEJUNUM

Case iv, under the heading of duodenal ulcer, illustrates the confusion in diagnosis in some of these cases.

Case x. Mr. R. A. D., aged sixty-eight, who forty years ago had several attacks of pain, nausea, occasional vomiting, gas and soft stools, which was empirically diagnosed as bilious colic and for which he was given calomel and salines and relieved. During such an attack eighteen years ago, under the diagnosis of appendicitis an exploratory operation was advised and halted by his surgeon and friend Dr. John Gibbon, who saw him in consultation with New York surgeons

and advised a wait on account of atypical symptoms and uncertainty of diagnosis. To this day he occasionally has a little nausea and soft stools, quickly relieved by an oil purge. His physical findings, except for slight tenderness over the epigastrium during an attack, are negative. *X*-ray studies show a diverticulum involving the second portion of the duodenum, which is smooth in outline, freely movable, not tender to palpation and the barium meal is seen to enter and leave the diverticulum during fluoroscopic examination (Fig. 12). The six-hour picture shows a small residue in the diverticulum after the stomach and duodenum have emptied (cf. Figs. 13 and 14, Cases XI and XII).

Fig. 8. Case VII. Markedly dilated duodenum after gastroenterostomy, so called vicious circle, followed by persistent duodenal obstruction. Completely relieved by duodenojejunostomy at second operation.

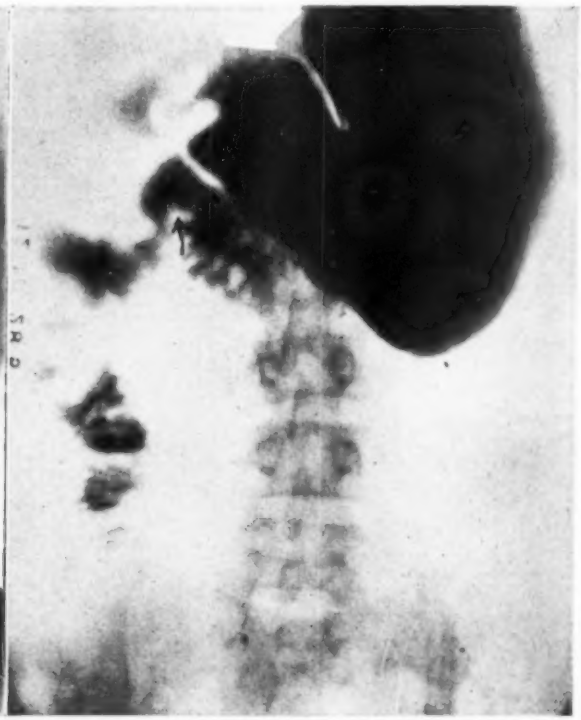

Fig. 9. Case VII. Rapidly emptying duodenum after duodenojejunostomy.

Case XIII. Mrs. McQ., aged fifty-two, 65 lb. loss of weight, slight gastroenteroptosis, pain and distress felt in epigastrium, nausea, attacks of vomiting and soft stools. Moderately dilated duodenum between attacks. Diverticula not found in roentgenograms, visualization of gall bladder negative. Operated on for prolapsus uteri with exploration of upper abdomen. Two diverticula found just distal to duodenojejunal function, one size of lemon with all coats of the intestine and abundant separate blood supply from that of intestine with vessels branching separately from both layers of mesentery.

This diverticulum resembled largest Meckel's diverticulum I have seen, with a blood supply suggesting a congenital origin. The second diverticulum was small, thin walled and no such blood supply (Cases XI and XII, Figs. 13 and 14). The large diverticulum by location, adhesion and weight, when filled produced an intermittent and partial obstruction at the duodenojejunal angle and produced the attacks of nausea and vomiting and partial obstruction of the duodenum.

E. Compression of Duodenum by Mesenteric Pedicle of Superior Mesenteric and Median Colic Arteries (Duodenal Arteriomesenteric Ileus)

Case XIV. Mrs. B., aged forty-two, attacks of pain in epigastrium as well as pain in right lower abdomen, profuse menstruation, vomiting, elevation of temperature and leucocytosis, negative urine. Poor result with enemata. Brought to hospital as acute abdominal case and diagnosis of ovarian cyst. Was formerly

operated on for tuberculous spine with bone graft and appendectomy. Abdomen flat, scar of former appendectomy, tenderness and definite resistence over cecal region. Soft mass over right adnexa, small mass over left adnexa. Diagnosis: Ovarian cysts right and left, partial intestinal obstruction. At oprateion ovarian cyst size of lemon on left side. Cecum down in right side of pelvis, filled with soft doughy fecal matter, ascending colon adherent to peritoneum, at site of former appendectomy. Colon liberated from abdominal wall, enucleation of left ovarian cyst. Patient vomited after operation large quantities of bile-stained fluid, in spite of good results following enemata on the second and third days, and continued to vomit in spite of the fact that the abdomen remained soft. After a large enema and turning the patient over on her abdomen with foot of bed elevated, there was a cessation of vomiting and a sudden movement of bowels filling the bed. The patient was sustained on salt solution and glucose given in frequent and large quantities intravenously during her vomiting. By the fifth day she was entirely comfortable and taking food. Impression: Duodenal arteriomesenteric ileus (acute dilatation of stomach). Compare with Case VII (Figs. 8 and 9) and so-called vicious circle following gastroenterostomy.

FIG. 10. Case VIII. Slightly dilated duodenum. Duodenal obstruction relieved by lideration of adhesions and rolling of terminal duodenum. Operation of choice duodenojejunostomy to prevent recurrence of adhesions.

FIG. 11. Case IX. Gastroenterostomy between detached duodenojejunal angle and stomach. Extremely rapidly emptying stomach, vomiting and colitis. Projected operation: Closure of gastroenterostomy and duodenojejunostomy.

CASE XV. Mr. D., aged thirty-one. Attacks of pain in epigastrium, at times very violent, followed by frequent and voluminous vomiting of bile-stained fluid. Pale, loss of weight and weakness. Attacks, at first, spaced by months' time, but became more frequent. This case was followed over a long period with repeated *x*-ray studies between attacks, which studies showed a gastroenteroptosis with moderate dilatation of duodenum. Finally examined during an attack; posture failed to relieve him. Barium meals given him were vomited. By persistent giving of barium while under the fluoroscope, the duodenum was seen to dilate widely, peristalsis immediately became very active, a small amount of barium would pass the terminal duodenum, quickly followed by violent antiperistalsis and rapid back-flow into the stomach, contraction of the stomach

and vomiting. After many attempts a film was obtained (Fig. 15). At operation, the duodenum was dilated, there was a rolling finding and employing definite fibers of crystallization as described under the nephrocolic ligament of Longyear. This operation was

Fig. 12. Case x. (Cf. Figs. 13 and 14). Large diverticulum of second portion of duodenum. Occasional pain and nausea, followed by diarrhea. Not operated on.

Fig. 13. Case xi. Diverticula in second portion of duodenum and in terminal duodenum. No obstruction, minor symptoms, not operated on. Similar case showed duodenal obstruction. Relieved by liberation of adhesions and resection of diverticula.

of the terminal duodenum on the peritoneum with a few light adhesions, the mesenteric pedicle of the superior mesenteric artery pressed tightly down on the terminal duodenum and when lifted with the finger under it, could be felt to pull down on the finger with considerable weight. An inframesocolic duodenojejunostomy was done. The patient has experienced a complete relief of pain and vomiting immediately from time of operation to date, with absolute feeling of well-being and an appreciable gain in weight and color. Although but three months following operation his gain is still progressive.

Case xvi. Miss McL., aged twenty-eight. Pain over right back, right lower abdomen, attacks of violent vomiting. The right kidney felt in the region of the cecum. *X*-ray studies showed the cecum in the location of the pelvis. Diagnosis: Nephrocoloptosis. The kidney was fixed after the method described by Longyear, done ten years ago, the kidney remains in position, the urine is negative and the cecum occupies about its usual position. There have been no more attacks of vomiting, the patient is in good health and comfort except for slight pain in the right lumbar region. Impression: Duodenal stasis form dragging on duodenum and compression by mesenteric pedicle of median colic artery.

Case xvii. Miss T., aged twenty-three. Very thin, pale girl, complaining of marked constipation and great difficulty in emptying bowels, weakness; even talking is great effort and voice is almost lost. Gastroenteroptotic type, enormously dilated cecum and ascending colon entirely prolapsed into pelvis. Attacks of pain in epigastrium and continuous voluminous vomiting. At operation an enormously dilated cecum and ascending colon, six inches across transverse diameter with thickened

walls and filled with doughy fecal matter was lifted out of pelvis and brought entirely out of incision. There was no parietal peritoneal attachment until the hepatic flexure was reached and the attachment here was a long loose membrane. The mesocolon was easily tied and severed and the entire cecum and ascending colon resected at the hepatic flexure, where a lateral anastomosis was done between the ileum and transverse colon, just distal to the hepatic flexure. There have been no more attacks of pain in the epigastrium and vomiting, but a gain in weight, strength and color and a return of a strong voice. The girl has been working for two months following her operation and has applied to the hospital for nurse's training. *X*-ray studies following operation show a ready passage of barium through stomach, duodenum and transverse colon with delay in descending colon and sigmoid. She still requires petrolagar for abundant bowel movements. Impression: Duodenal stasis from compression by mesenteric pedicle of median colic artery.

Fig. 14. Case xii. Diverticulum of upper jejunum, rarely shown in *x*-ray studies. Diverticula in case occurred in upper jejunum as well as at duodenojejunal junction. (Cf. Fig. 13.)

Fig. 15. Case xv. Marked recurrent dilatation of first and second portions of duodenum becoming more fixed. Compression by arterial pedicle of superior mesenteric artery and adhesions. Relieved by duodenojejunostomy.

DUODENAL OBSTRUCTION

In the seventeen cited cases there will be noted that pain, attacks of vomiting, loss of weight and color were the main characteristics in 11, namely Cases iii, v, vi, vii, viii, ix, xiii, xiv, xv, xvi, xvii, and to a milder degree in Cases i and ii. Cases i and ii were both of the gastroeneteroptotic type, both vomited blood. *X*-ray studies showed the ulcer and a dilated duodenum. Case i had a recurrence of ulcer and perforation after closure of second ulcer and gastroenterostomy; Case ii showed ulcer below the duodenal cap and was relieved by gastroenterostomy. It was suggested that duodenojejunostomy followed by resection of the ulcer should have been the operation of choice in both cases, to drain more directly the dilated duodenum and would likely have prevented the recurrence of ulcer and perforation in Case i and have secured more permanent results in Case ii. Case ix was overdone and the effect produced was that of combined duodenojejunostomy and gastroenterostomy. Case vii was a failure after gastroentero-

stomy and was relieved after additional duodenojejunostomy. Both cases should have received a duodenojejunostomy only, at the first operation. Case III was frankly cholecystitis and cholelithiasis, but even in this case the gall bladder was intimately adhered to the duodenum, producing the artefact of an ulcer. In only 1 of the 5 cases of diverticula, Cases IV, X, XI, XII, XIII, were pain and vomiting prominent symptoms and in Case XIII the diverticulum near the duodenojejunal angle so lay and adhered as to obstruct the duodenum; symptoms in Case IV were likely due to the gall bladder since it failed to visualize after the dye and two *x*-ray studies. Cases VII and XIV were temporarily relieved of pain and voluminous vomiting by posture; Case VII was permanently relieved at the second operation by duodenojejunostomy; Case VII developed the so-called vivious circle following gastroenterostomy and was later relieved at second operation by duodenojejunostomy. Case XIV developed the so-called acute dilatation of the stomach following the operation on the lower abdomen. Duodenal obstruction, vicious circle and acute dilatation of the stomach are frequently most likely one and the same thing. In Cases XVI and XVII the drag and pull and hence compression of the duodenum by the mesenteric pedicle of the median colic artery were relieved by nephrocolopexy (Longyear) and resection of megalocecum and colon respectively.

By reconstruction of clinical findings and *x*-ray studies in these cases of duodenal obstruction, the following symptoms are to be noted as most prominent and characteristic:

1. Vomiting of bile-stained fluid often in enormous quantities.

2. Pain located over the whole epigastrium or right hypochondrium, sensation of weight and discomfort or the negro's "misery" in the epigastrium, between attacks and violent pain during attacks.

3. Emaciation and pallor; the gastroenteroptotic type of patient.

4. Relief by posture or abdominal supports except when the obstruction has become fixed, as in Case VII, so-called vicious circle and in Case XIV so-called acute dilatation of the stomach or by taking away the weight on the mesenteric pedicle of the median colic and superior mesenteric arteries by nephrocolopexy or resection of the colon as in Cases XVI and XVII.

5. The duodenogastric type, severe and with acute duodenal obstruction, in which the organic breakdown pushed to a maximum and death may ensue, Cases V–VII.

The colonic type with right-sided constipation and cecocolonic stercoral stasis dominates the scene with duodenal compression as an accessory, Cases XIV, XVI, and XVII.

6. Complications, particularly of duodenal ulcer, recurrent duodenal ulcer and duodenal ulcer below the cap, Cases I and II.

7. *X*-ray findings of dilated duodenum, gastroenteroptosis, coloptosis studied during an attack of pain and vomiting, as Case XV; marked dilatation of the duodenum stopping suddenly at the midline; antiperistalsis and churning movements in the distended duodenum, reflux into the stomach, antiperistalsis in stomach, contraction and vomiting or antiperistalsis passing up duodenum and stomach at the same time with projectile vomiting.

There is a sharp line of demarkation between the right part of the third portion of the duodenum filled with the opaque meal and the left part which is empty, Case XV (Fig. 15).

TREATMENT

The procedures employed in 13 of the 17 cases cited here were:

1. Posture in so-called acute dilatation of the stomach following operation on the lower abdomen, Case XIV and in so-called vicious circle following gastroenterostomy in Case VII with temporary relief in both cases.

2. Simple liberation of terminal duodenum, Case VIII.

3. Gastroenterostomy, Case VII and anastomosis of duodenojejunal angle, equal to gastroenterostomy plus duodenojejunostomy, Case IX with failure in both cases. Duodenojejunostomy alone would likely have been the proper original procedure, likewise gastroenterostomy as final operation in Cases I and II when duodenojejunostomy would have been better surgical judgment, although both cases are relieved of symptoms.

4. Nephrocolopexy, Case XVI, relieved for ten years.

5. Cecocolectomy, Case XVII relieved for eighteen months.

6. Resection of diverticulum of jejunum and liberation of adhesions, Case XIII.

7. Duodenojejunostomy, Cases V, VI, VII and XV with relief of symptoms immediately and permanently and with results equal to the relief of any case of acute high intestinal obstruction.

X-ray studies following duodenojejunostomy show the opening functioning perfectly, that the duodenum emptying is regular and that antiperistalsis has disappeared, Case VII (Figs. 8 and 9).

The operation of duodenojejunostomy is the operation of choice in the duodenogastric type of duodenal obstruction, in certain cases of duodenal ulcer with gastroenteroptosis and dilated duodenum and in all of acute duodenal obstruction, regardless of cause of obstruction, where and when, on account of time, remote causes of obstruction cannot be determined.

LYMPHANGIOMA OF THE MESENTERY

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BENIGN tumors of the mesentery, judging from the infrequent case reports in the literature, are of comparatively rare occurrence. The first case of mesenteric tumor successfully treated by operative procedure was reported by Tillaux in 1880. Since then, cases have been reported in the literature from time to time, most of them being sarcomas, fibromas or lipomas. Lymphangioma or lymphoma is apparently of exceedingly uncommon occurrence. Harris and Herzog[1] have reviewed 57 cases of solid mesenteric tumor, only 1 of which was of lymphomatous nature. Begouin[2] collected 29 cases and Vance[3] 27 cases, none of which were classified as lymphangioma. In a review of a series of cases of mesenteric tumor, Bowers[4] stated that of the solid benign tumors of the mesentery, lipomas predominate, with fibromas being of rather infrequent occurrence. He did not mention tumors of lymphoblastic origin. Royster[5] has reported a case of blood tumor of the mesentery, which may have been a hemorrhagic lymphangioma. Bigelow and Forman[6] cite a case of lymphosarcoma of the mesentery. Ransohoff and Friedlander[7] state that mesenteric tumors are almost invariably of connective tissue origin, the vast majority being fibromas, fibromyxomas, lipomas or sarcomas. Nassen,[8] in his series of cases, reported 1 case of lymphangioma of the mesentery of the ileum in a man aged forty-one. Extirpation of the tumor was performed with complete recovery. Rawls[9] published the report of a case of lymphangioma of the mesentery of the ileum occurring in a boy aged thirteen. The tumor was excised with an excellent result. Westman,[10] in an admirable article on mesenteric lymphangioma, reported a case in which the tumor was found in the mesosigmoid of a boy aged eleven. Removal of the growth was followed by uneventful recovery. Judd[11] removed a lymphangiolymphoma from the mesentery of the jejunum in a woman aged twenty-eight with an excellent result.

CASE REPORT

The patient, a man aged thirty-nine, by occupation a clerk, came under observation November 15, 1928, complaining of intermittent dull pain in the lower abdomen, of about six months' duration. The pain apparently seemed localized in the lower midabdomen, dull, nagging in character, never severe, never crampy, and did not radiate. The patient noticed the pain especially when he was bending over his desk. About two weeks previous to observation, he thought he had felt a lump in his lower abdomen while he was bathing. He had no other abdominal symptoms. General health was excellent, no digestive disturbance, no loss of weight. The family history was non-essential. Aside from a few childhood diseases, the past history was without incident.

The patient was a well-developed, well-nourished man weighing 164 lb. In the lower mid-abdomen there was an easily palpable, firm, rounded mass, about the size of a hen's egg. The mass was not tender, and was exceedingly mobile, being easily pushed over to both the right and left lower abdominal quadrants. There was no demonstrable regional adenopathy. Rectal examination was negative. The rest of the physical examination was essentially negative. The hemoglobin was 82 per cent, erythrocytes 5,100,000 and leucocytes 6400. The differential count was normal. The blood Wassermann test and urinalysis were negative. Roentgenograms of the chest showed no evidence of metastasis. A colon ray with barium enema was negative. A clinical diagnosis was made of abdominal tumor, origin indeterminate, and exploration advised.

The abdomen was opened by a mid-right rectus incision. On exploration the mass was found to be a rather smooth, solid tumor,

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situated in the mesentery of the ileum about 15 cm. from the ileocecal juncture. Fortunately it was far enough away from the bowel so that it could be shelled out from between the mesenteric leaves without interfering with the blood supply to the gut. A few of the smaller mesenteric vessels required tying and the hole in the mesentery was closed with plain catgut. Further abdominal exploration was negative. As the color of the bowel adjacent to the mesenteric repair remained normal, the abdomen was closed. Convalescence was uneventful. The pathological report was: Tumor composed mostly of spongy lymphangiomatous tissue, with some areas of lymphoid tissue, the whole enclosed in a fibrous capsule. No evidence of malignancy. Diagnosis: Lymphangioma of the mesentery.

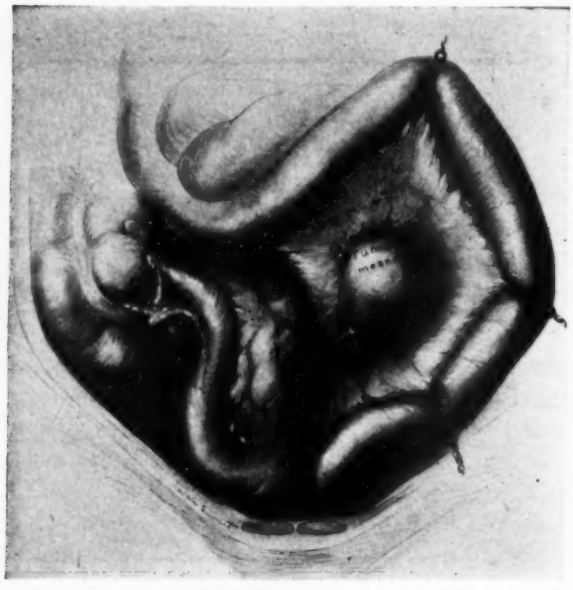

FIG. 1.

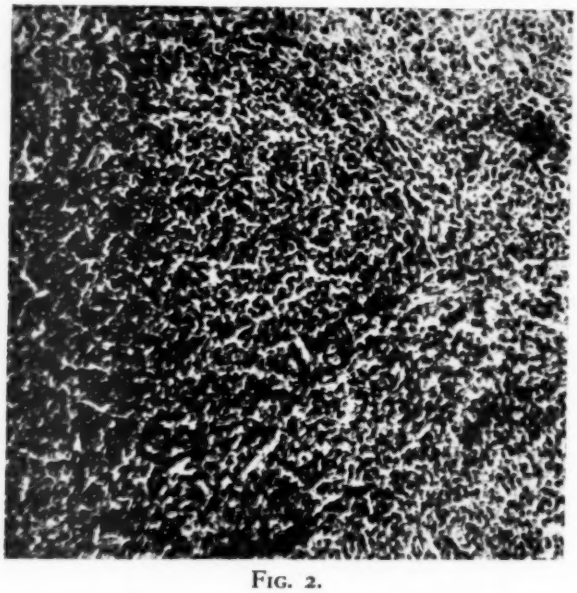

FIG. 2.

COMMENT

In regard to the structure of these lymphoblastic mesenteric growths it may be said that they are mainly composed of lymphangiomatous tissue in conjunction with typical lymph-glandular tissue. They are thus probably true lymphangio-lymphomas. The pathogenesis of these tumors is, of course, a matter of conjecture. The earlier investigators seem to favor the conception that lymph stasis with consequent dilatation of the lymph vessels is a cause of the more cavernous spongy-like lymphangiomas. The theory has been advanced that this stasis might be caused by the blocking of lymph channels by inspissated chyle, with accompanying degeneration of adjacent lymph nodes. The presence of lymph-glandular tissue in most of these tumors has been used as an argument in favor of this conception. Westman very logically rejects the stasis theory on the grounds that (1) the numerous anastomoses in the lymphatic system would take care of any stasis through collateral circulation, and (2) it is not always possible to determine any anatomic-pathologic changes that might give rise to stasis. Recent investigators, notably Westman and others who have extensively studied this subject, apparently hold to the idea that these tumors are true lymphatic growths, possible of congenital origin.

Pathological examination of the specimen in our case showed the tumor not only to be composed of typical spongy lymphangiomatous tissue, but also in part to have a solid lymph-glandular structure. The presence of lymphoid tissue in certain organs undergoing pathologic change is not uncommon. Notable examples of this

are found in cases of chronic thyroiditis, where practically the entire thyroid gland is often found to be replaced by true lymph-glandular tissue, with typical germ centers showing mitotic figures, lymphoblasts and lymphocytes. Cases of Mickulicz' disease of the submaxillary gland and Kuttner's disease of the lacrimal gland also show the same presence of lymphoid tissue. Hashimoto[12] has demonstrated the presence of true lymphoid tissue in certain cases of a chronic inflammatory process in the gastric mucosa. Whether this lymphoid tissue is the result of chronic inflammation, or is simply a new growth it is impossible to say. It seems not improbable that lymphangioma of the mesentery would fall in the same category as these other lymphoid formations.

The clinical diagnosis of mesenteric tumor is practically an impossibility. There are no pathognomonic signs or symptoms. All writers agree that the abnormal mobility of the tumor, when palpated through the abdominal wall, is the only fairly distinctive sign. It is stated that no abdominal tumor possesses the wide range of motion that mesenteric tumors do. This was certainly a striking feature in our case, it being possible to move the tumor through both the lower abdominal quadrants by palpation.

In the differential diagnosis one must consider mainly ovarian cyst, enlargement and displacement of the kidney, pedunculated fibromyoma, omental cyst, tumor of the bowel itself and pancreatic cyst.

Surgical exploration of the abdomen with the extirpation of the tumor is the procedure of choice. The mortality following operation as reported in the literature is high.

Begouin showed a mortality of 51.7 per cent in 29 collected cases, Vance 40.7 per cent in 27 cases, and Rawls 45.1 per cent in 13 cases. The necessity of intestinal resection greatly increases the risk. Injury to the mesenteric vessels with consequent interference with the circulation of the adjacent segment of bowel must be carefully avoided.

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SARCOMA OF THE STOMACH

REPORT OF FOUR CASES AND REVIEW OF THE LITERATURE*

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MALIGNANT new growths of the stomach other than those of an epithelial nature are relatively uncommon enough to be of interest to both the clinician and the pathologist. Our recent observation of 2 cases of gastric sarcomas served to awaken our interest in the tumor, and has led us to examine the records of the Charity Hospital of New Orleans, as well as to an extensive survey of the available literature on the subject. It is the purpose to here record our cases, and to present a discussion of the salient features of the condition, based principally on our survey of the literature.

CASE REPORTS

CASE I. L. R. (C. H. 363–1) Negro female, aged thirty-five years, was admitted with the complaint of pain in the stomach of one year's duration. Past history negative. Physical examination revealed emaciation and a palpable epigastric mass. Laboratory examinations negative. The *x*-ray diagnosis was a "partial pyloric stenosis of malignant origin." At operation the pyloric end of the stomach was found to be diffusely thickened to an average thickness of 2 cm. with marked pyloric stenosis. There were dense adhesions to the pancreas and the regional lymph nodes were enlarged. The stomach was explored and closed, and a gland removed for histological study. The report of the pathologist on this tissue was sarcoma. The patient made an uneventful postoperative recovery and was dismissed from the hospital a few weeks later. No follow-up of the case was possible.

CASE II. R. F. (C. H. B7329) Negro female, aged fifty-six, was admitted with the complaint of a "lump in the stomach," which she had noted about a year ago. Physical examination revealed a palpable epigastric mass. At operation a large vascular tumor with many adhesions to the surrounding viscera was found springing from the greater curvature. The tumor was resected and a gastrorrhaphy performed. The tumor weighed 850 gm. The report of the pathologist was "malignant leiomyoma with much hyalin degeneration." The patient made an uneventful postoperative recovery. Further developments in the case are unknown.

CASE III. J. R. (C. H. 633) white, male, aged sixty, was admitted with the complaint of severe epigastric pain of about eight months' duration. Physical examination revealed marked emaciation, and enlargement of the cervical and inguinal lymph nodes. The *x*-ray reported that there was a filling defect in the prepyloric portion of the stomach. At operation a dense infiltrating growth was found at the pylorus, which grossly appeared to be a scirrhous carcinoma. There was a mass of enlarged glands of dense consistency in the mesentery of the jejunum. A partial gastrectomy and a gastrojejunostomy were done. The patient died a few days later. The report of the pathologist was "round cell sarcoma."

CASE IV. M. H., aged fifty-six years, suffered from indigestion for the past year, accompanied by loss of weight. *X*-ray examination suggested an intragastric growth. At operation a tumor mass 800 gm. in weight and attached to the lesser curvature was found. Histological examination revealed it to be a spindle-cell sarcoma. The patient is in perfect health four years later. (Figs. 1 and 2.)

HISTORICAL REVIEW

In 1847 Bruch recorded the first case of sarcoma of the stomach. Forty years later, Virchow reported on the first patient that came for operation. In 1900 Fenwick was able to gather 60 reported cases, and decided that 53 of these were adequately proved. In the following decade critical studies and case reports were made by Lecene and Petit, Corner and Fairbanks, Lafero, Azzurini and others. In 1914 Flebbe collected 157 cases based solely

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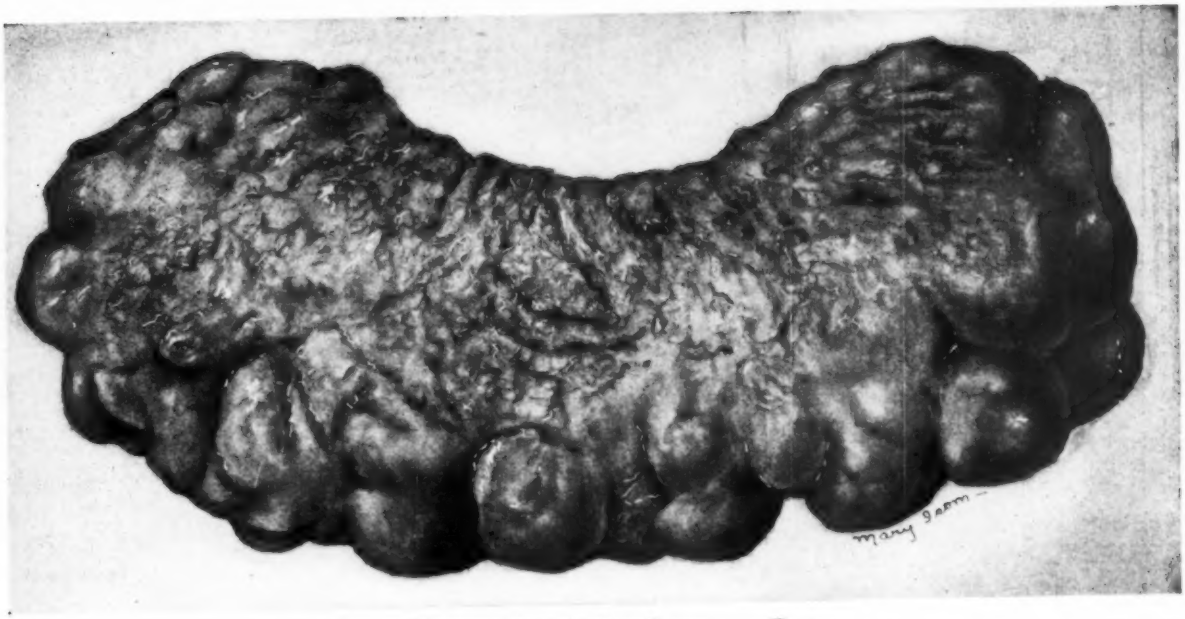

FIG. 1. Intragastric spindle-cell sarcoma. Case IV.

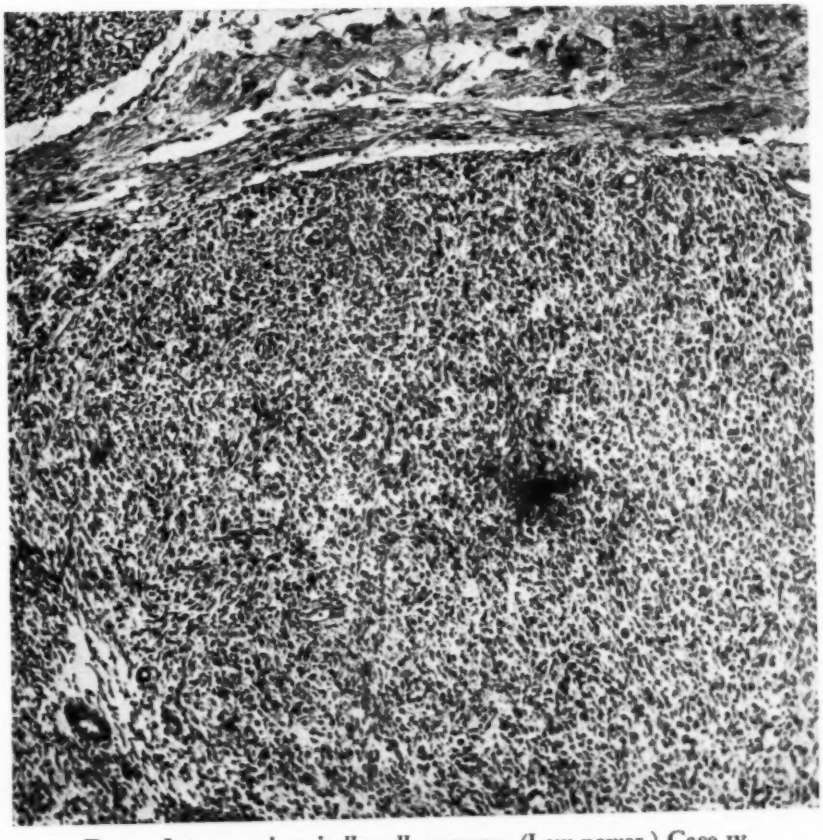

FIG. 2. Intragastric spindle-cell sarcoma. (Low power.) Case IV.

TABLE I

Author & Year	Age	Sex	Main Symptoms	Duration	Operation	Location of Tumor	Metastases & Recurrence	Histology of Tumor	Comments
Krichebski, S. A., 1913..... Case I	21	M.	Abdominal pain & tumor. Diarrhea. Coffee-ground vomitus.	1 year	.....	Diffuse in lower ⅗ stomach wall.	Pancreas.	Round-cell	Case I. Nine days after admission to hospital died suddenly. Nooperation—autopsy.
Case II.....	36	M.	Epigastric tumor.	4 months	Resection Billroth II.	Pylorus.	Pancreas.	Round-cell.	Case II. Collapsed and died 3 days after operation. No autopsy.
Scudder, C., 1913.....	29	M.	Indigestion.	2 years	Partial gastrectomy with preliminary jejunostomy.	Endogastric posterior wall.	.....	Spindle-cell.	Reported as well 6 months after operation.
Gossage & Hicks, 1913.....	37	M.	Abdominal pain dizziness, bloody vomitus & bloody stools.	17 days	.....	Attached by narrow pedicle to middle of posterior wall.	.....	Fibrosarcoma.	Death several days after admission to hospital. Autopsy.
Schiller, M., 1914.....	67	M.	Palpable abdominal tumor.	9 years	.....	Not stated.	.....	Leiomyosarcoma.	The interesting thing about this case was the existence of a palpable tumor for 9 years. It may have been a myoma which became sarcomatous.
Kimpton, A. R., 1914.....	30	F.	Palpable abdominal tumor. Indigestion.	20 years	Partial resection.	Not stated.	Regional lymph nodes.	Round-cell.	Living and well at time of report, 6 years after operation. A later report in 1919 states that patient was still living. That this was originally a leiomyoma which developed malignant properties, is suggested by the long duration of the tumor.
Frazier, C. H., 1914.....	36	F.	Vomiting & heartburn. Palpable abdominal tumor.	About 1 yr.	Gastric resection & posterior gastrojejunostomy.	Greater curvature.	Jejunum. Regional lymph nodes.	Lymphosarcoma.	Well 9 months later.
Hartz, H., 1914.....	41	M.	Indigestion pain in stomach. Nausea.	2 months	Exploratory laparotomy. Biopsy of omentum.	Anterior wall lesser curvature.	Gastrocoli omentum & head of pancreas.	Small round cell.	At operation inoperable tumor found, biopsy of metastasis to omentum for diagnosis. Death 16 days later. Autopsy.
diGiacoma, G., 1914..... Case I	65	M.	No clinical signs.	.....	.....	Near pylorus.	.....	Round-cell.	Case I. Discovered at autopsy on patient dying of pulmonary

									tuberculosis. Chronic polypous gastritis present.
Case II	91	F.	No clinical signs.	.....	.....	Pyloric region.	.....	Small round-cell.	Case II. Discovered at autopsy on patient dying of cerebral apoplexy.
Storch, B., 1914. Case I	40	F.	Dyspepsia. Epigastric pain. Palpable epigastric tumor.	1 year	Resection Billroth II	Posterior surface lesser curvature.	Glands in gastrocolic ligament recurrence at site.	Round-cell.	Case I. Returned 9 months after operation with large recurrence at site and signs of metastases tases to liver. Dismissed as inoperable.
Case II	41	M.	Loss of weight and appetite bloody stools.	6 months	Retrocolic posterior gastroenterostomy.	Greater curvature near pylorus.	Liver.	Round-cell.	Case II. Developed fistula at operative site. Died about 6 months later. Autopsy.
Case III	58	M.	Pain in stomach.	3 weeks.	Resection Billroth II.	Posterior wall near pylorus.	Glands in gastrohepatic ligaments.		Case III. The stomach wall also contained a benign mucous membrane fibroids operated about 6 months previous to report, and no mention made other than "uneventful recovery."
Case IV	37	M.	Loss of appetite constipation, epigastric tumor.	6 months.	Exploratory laporotomy.	Location in stomach not given.	Peritoneum intestine, liver.	Lymphosarcoma.	Case IV. Died about 2 weeks after exploratory laporotomy. Autopsy. A clinical diagnosis of tuberculous peritonitis had been made.
Burty, 1914	58	F.	Palpable abdominal tumor.	5 months.	Resection of circular disc of stomach wall around pedicle.	Attached by pedicle to lower part of greater curvature.	.....	Angiosarcoma.	Uneventful recovery, and well at time of report, about 6 months after operation.
(Note cases reported by diGiacoma in 1915 were same as those reported in 1914.)									
Schlesinger, H., 1915	17	F.	Vomiting & other symptoms of pyloric stenosis.	6 months.	Resection.	Pylorus.	.....	Lymphosarcoma.	An unusual feature of this case was the fact that it produced a pyloric stenosis, which sarcomas, particularly of this type seldom do.
Venturelli, G., 1915	71	F.	Abdominal pain, dizziness.	1 month.	.....	Greater curvature.	Omentum.	Lymphosarcoma.	Patient stated that her symptoms dated from a blow which she received in her left flank.
Scott & Forman, 1916	53	M.	Weakness & abdominal pain.	.....	Exploratory laporotomy. Removal of glands	General infiltration of stomach wall.	Regional lymph nodes.	Malignant lymphoblastoma.	Died 2 days postoperative. This case was autopsied and

TABLE I (*Continued*)

Author & Year	Age	Sex	Main Symptoms	Duration	Operation	Location of Tumor	Metastases & Recurrence	Histology of Tumor	Comments
Scott & Forman, 1916 (*Continued*)					for diagnosis.				primary neoplastic process was found to be in stomach. It is reported as a case of Hodgkin's disease of stomach. Pathologists report on tissue removed at laparotomy was "malignant lymphoblastoma."
Barington-Ward & Shaw, 1916.....	25	M.	Indigestion.	5–6 years.	Resection partial gastrectomy.	Pedunculated attached to anterior & posterior walls near pylorus.	None seen.	Round-cell.	Well at time of report 4 months after operation.
Bachrach, R., 1916.....	39	F.	Nodule in gastric region, later became mass.	12 years.	Resection of portion stomach wall where tumor attached.	Pedunculated attached at upper pole & greater curvature.	.....	Malignant myoma.	Uneventful recovery dismissed as cured 14 days after operation. Discussion of the possibility of this tumor having had its start as a benign growth which underwent sarcomatous changes.
Schlesinger, H., 1916.....	32	M.	Gastric pain vomiting of solid food, symptoms occurred only occasionally.	7 years.	.....	Lesser curvature, posterior wall.	Liver, spleen left kidney.	Lymphosarcoma.	Discovered at autopsy.
Case II.....	66	M.	Loss of appetite emaciation, vomiting.	18 months.	Exploratory laparotomy.	Greater curvature central between cardia and pylorus.	Lymphatic glands of lesser curvature, liver left suprarenal.	Lymphosarcoma.	Case II. At laparotomy found inoperable. Died a few days later. Autopsy.
Saito, J., 1916.....	27	F.	No definite clinical symptoms.	.....	.....	Lesser curvature from cardia to pylorus.	.....	Carcinosarcoma.	Discovered at autopsy. Author states that the cases of Queckenstedt (1904) Lippmann (1904) & Lindemann (1908) were of the same histological type as his own.
Rhodenburg, G., 1917.....	No details reported			.....	.....	.....	.....	Fibromyxosarcoma.	
Medina & Egana, 1917.....	37	M.	Epigastric pain.	6 months.	Gastric resection.	Lesser curvature.	.....	Called round-cell sarcoma by 3 pathologists, and fibrosarcoma by one.	

Geymuller, E., 1917	40	F.	Epigastric pain.	1 year.	Gastric resection.	Prepyloric region.	.....	Fibrosarcoma.	
Fritzsche, R., 1918	39	M.	Occasional epigastric pain, bloody vomitus, and bloody stools.	3 years.	Laporotomy at times of perforation. Drainage of peritoneal cavity.	.....	.....	Leiomyosarcoma.	Death 9 days after operation. Autopsy. Perforation with death due to peritonitis. Question of malignant degeneration of benign myoma.
Schroeder, R., 1918	Details of case not obtained								
Pagenstecher, G. A., 1919	43	F.	Rapidly growing abdominal mass.	1 month.	Exploratory laporotomy.	Anterior wall.	.....	Myosarcoma	Died 12 days after operation. Autopsy established diagnosis.
Holmes, T. W., 1919	48	M.	Sense of oppresion in epigastric.	1 month.	Resection.	Greater curvature.	.....	Fibrosarcoma.	Living and well 6 years later.
McWhorten, G. L., 1919	45	M.	Abdominal swelling.	7 months.	Exploratory laporotomy with removal of 2500 c.c. clear fluid.	Greater curvature.	.....	Round-cell & spindle-cell sarcoma.	Died 10 days after operation. Autopsy. No metastases noted.
Koettlitz, H., 1919	31	M.	Abdominal tumor. Indigestion.	1½ years.	Inoperable tumor discovered at exploratory laporotomy. Tissue for diagnosis removed.	Generalized infiltration in all but cardiac portion.	Mesentery pancreas.	Fibrosarcoma.	Died 1 month after operation.
Loeper, M., 1919	?	M.	Abdominal tumor and pain. Vomiting.	2 months.	.....	Anterior wall.	Omentum & regional lymph nodes.	Round-cell.	Died 4 months after onset symptoms.
Noordenbos, 1919	17	F.	Recurrent hemorrhage.	3 years.	Subtotal gastric resection.	Lesser curvature	.....	Spindle-cell.	Good postoperative recovery.
Brasch, S., 1920	22	F.	Gastric distress.	1 year.	Complete resection.	Pyloric end.	.....	Large round-cell.	Condition good 15 months later.
Douglas, J., 1920 Case I	38	F.	Mass in abdomen pain.	.....	Resection pyloric end.	Pyloric end greater curvature.	Regional lymph nodes.	Lymphosarcoma.	Died 24 hours after operation.
Case II	41	F.	Epigastric pain.	2 years.	Resection pyloric end.	Pyloric end.	.....	Lymphosarcoma.	Died 23 days after operation.
Case III	36	M.	Epigastric pain.	3 months.	Resection.	Posterior wall.	.....	Lymphosarcoma.	Well 16 months later.
Haggard, W. D., 1920 Author's case	17	F.	Mass in abdomen hemorrhage.	1 year.	.....	Lesser curvature.	.....	Fibromyosarcoma.	Well at time of report 20 months after operation.
Reports following 13 cases from Mayo Clinic—1908 to Aug. 1920.									
1. Case 18165	38	F.	Abdominal tumor.	3 years.	Partial gastrectomy.	Greater curvature.	.....	Fibrosarcoma.	Died 6 months later.
2. Case 53972	62	M.	Epigastric pain.	4 years.	Explorotory laporotomy. Removal of gland.	Fundus.	"Extensive metastases."	Sarcoma.	Condition "poor" 4 months after operation.
3. Case 82074	44	.....	Epigastric pain abdominal tumor.	4 weeks.	Resection.	Greater curvature.	.....	Spindle-cell.	Well 4 months later.
4. Case 182060	16	M.	Continuous epigastric pain.	4 months.	Partial gastrectomy.	Posterior wall.	.....	Lymphosarcoma.	Development unknown.

TABLE 1 (*Continued*)

Author & Year	Age	Sex	Main Symptoms	Duration	Operation	Location of Tumor	Metastases & Recurrences	Histology of Tumor	Comments
5. Case 188936	62	M.	Epigastric pain.	6 months.	Partial gastrectomy.	Lesser curvature & pylorus.	.....	Lymphosarcoma	Development unknown.
6. Case 304520	44	F.	Epigastric pain.	7 years.	Polya resection.	Posterior wall.	.....	Myosarcoma.	Development unknown.
7. Case 205800	53	M.	Indigestion.	30 years.	Polya resection.	Pylorus.	.....	Lymphosarcoma.	Died 4 months later.
8. Case 266434	42	M.	Gastric distress.	4 months.	Subtotal gastrectomy.	Lesser curvature.	.....	Lymphosarcoma.	Died 3 months later.
9. Case 282682	66	M.	Weakness vomiting blood.	2 years.	Partial gastrectomy.	Lesser curvature.	.....	Mixed-cell sarcoma.	Well 9 months later.
10. Case 309588	40	F.	Abdominal pain nausea.	1 year.	Partial gastrectomy.	Pylorus.	.....	Myosarcoma.	"Good immediate recovery."
11. Case 289467	62	F.	Epigastric pain.	8 weeks.	Partial gastrectomy.	Lesser curvature, posterior wall.	.....	Lymphosarcoma.	Died 7 days after operation.
12. Case 316187	47	M.	Indigestion.	1 year.	Partial gastrectomy.	Lesser curvature.	.....	Lymphosarcoma.	"Recovered."
13. Case 190100	46	M.	Pain in chest & vomiting.	.....	Partial gastrectomy.	Lesser curvature.	.....	Lymphosarcoma.	"Recovered."
Note—Seven of these cases (lymphosarcoma) will not be counted in the final tabulation as Broders & Mahle in 1921 reported these cases as lymphosarcoma from the Mayo Clinic.									
Klinkert, D., 1921	28	M.	Pyloric stenosis.	Not given.	Gastric resection.	Involvement of entire stomach with exception of cardia.	.....	Polymorphocellular.	"Operated with favorable results." This case is unusual in that it produced pyloric stenosis.
Lendon, A. A., 1921	37	M.	Anemia, bloody vomitus & stools.	.....	.....	Pyloric half of posterior wall, greater curvature.	.....	Round-cell & spindle-cell sarcoma.	Living and well 11 years later. This tumor was described as being imbedded in viscus "loosely attached and easily shelled out. Grossly appeared to be fibromyoma.
Morris, J., 1921	33	F.	Indigestion.	6 months.	Resection.	Lesser curvature pylorus.	.....	Round-cell.	Well 22 months later.

Broders, A., Mahle, A., 1921 — Report 12 cases of lymphosarcoma of the stomach seen at Mayo Clinic Jan. 1, 1913 to Jan. 1921.

Summary of histories:

1. Length of time of clinical symptoms ..... 6.8 months
2. Clinical symptoms.
   1. Loss of weight ..... 11 cases
   2. Pain ..... 9 cases
   3. Vomiting ..... 7 cases
   4. Bleeding ..... 2 cases
3. Average age ..... 46.25 year (youngest 16, oldest 62)
4. Sex ..... 11 males
5. Properative diagnosis:
   Carcinoma of stomach ..... 7 cases
   Ulcer of stomach ..... 1 case
   Abdominal tumor probably inflammatory ..... 1 case
   Malignancy of stomach ..... 1 case

Tumor of pancreas..... 1 case
Pyloric obstruction..... 1 case
6. Operations: 6 resections; 6 found inoperable at exploratory laporotomy.
7. Results:
2 resected cases died of peritonitis.
1 died 4 months later with recurrence.
1 died 5 months later with recurrence.
1 returned 7 months later with recurrence.
4 of 6 who had exploratories died 6 weeks to 4 months later.
Information regarding others not obtained 1 case.

Simon, 1921	Reports case of spindle-cell sarcoma of greater curvature. In discussion of same paper Marklewitz reports same type in female aged 65, no gastric symptoms, picture dominated by large liver metastases.								
Cutler, E., & Smith 1922. Case I.	59	M.	Nausea & weakness.	7 months.	Exploratory laporotomy.	Lesser curvature.	Regional lymph nodes. Diaphragm pericardium pancreas.	Lymphosarcoma.	Died shortly after exploratory. Autopsy.
Case II	57	M.	Epigastric tenderness.	7 months.	Resection.	Whole of stomach wall.	.....	Lymphosarcoma.	Well 1 year later at time of report.
Gillette, W. J., 1922	45	M.	Epigastric pain, bloody vomitus & stools.	3 months.	Resection.	Lesser curvature.	.....	Lymphosarcoma.	Well at time of report about 14 months later.
Finsterer, H., 1922	54	M.	Pain, vomiting abdominal tumor.	1½ years.	Resection.	Pyloric portion of stomach.	Liver.	Polymorphous cellular.	This tumor developed after an operation for eliminating duodenal ulcer about 2 years previously.
Kolbe, H., 1922	51	M.	Vomiting, abdominal tumor.	6 months.	.....	Pyloric region.	.....	Fibrosarcoma.	This tumor perforated 6 months prior to death producing an abscess of the abdominal wall which finally caused death by general peritonitis. Autopsy.
Schindler, R., 1922	60	F.	Vomiting, diarrhea.	Several months.	.....	Anterior wall.	Periaortic peripancreatic perigastric lymph nodes.	Lymphosarcoma.	Death. Autopsy. Between ages of 20 to 30 patient had signs and symptoms of gastric ulcers with hemorrhage and had been successfully treated for same.
Ruffin, R., 1923	60	M.	.....	.....	Resection.	Both walls & both curvatures.	Liver, pancreas, regional lymph nodes at hilum of left lung, recurrence at site.	Lymphosarcoma.	Death 5 months later with recurrence at site and metastases.
Pistocchi, G., 1923	50	M.	Asthemia, vomiting.	4 months.	.....	Greater curvature, anterior surface.	Liver.	Lymphosarcoma.	Death 5 months later. Symptoms dated from an abdominal trauma. Was this an etiological factor?
Charrier, A. & Riou, 1923	63	M.	Loss of weight, indigestion.	5 months.	Resection.	Anterior surface near pylorus.	.....	Malignant fibrochondroma.	Recovered from operation and dismissed in 18 days. Further results not given.

TABLE I (*Continued*)

Author & Year	Age	Sex	Main Symptoms	Duration	Operation	Location of Tumor	Metastases & Recurrences	Histology of Tumor	Comments
Harding, D. B., 1924	16	M.	Abdominal mass, pain.	4 months.	Resection.	Posterior wall.	.....	Lymphosarcoma.	Death 1 month later.
Lassila, V., 1924	69	F.	Abdominal tumor below umbilicus. Indefinite abdominal disturbances.	4 months.	Resection.	Posterior wall near pylorus.	.....	Small round-cell sarcoma.	Free from recurrence and well at end of 4 years.
Thatcher, H., 1924	77	M.	Weakness epigastric tumor.	3 months.	.....	Cardiac portion, greater curvature.	Liver.	Spindle-cell.	Death 3 days after admission to hospital autopsy.
Klaus, H., 1924	49	M.	.....	4 months.	Resection.	Lesser curvature, posterior wall.	.....	Myofibrosarcoma.	Well 1 year later.
Lindboe, E., 1924 Case I	36	M.	Indigestion, bloody stools.	2 years.	Surgical removal.	Posterior wall.	.....	Myofibrosarcoma.	Died 15 months later in an accident.
Case II	31	M.	Pain nausea vomiting.	Few months.	Polya resection.	Posterior side, distal half.	Recurrence, gland of greater curvature.	Round-cell.	Died of recurrence 6 months later.
Author mentions 3 other cases observed in Norway. No details save age and sex:									
Case III	Elderly	M.							
Case IV	37	F.							
Case V	57	F.							
Demel, R., 1924	8	F.	.....	.....	.....	Pedunculated on stomach.	.....	.....	This tumor perforated before resection.
Sebestyén, G. & Kaló, A., 1924	41	F.	Pain.	2 months.	.....	.....	Retroperitoneal lymph nodes.	.....	Death 5 months later.
Kapel, O.	38	F.	Abdominal tumor, melena, pain.	10 years.	Resection of stomach & transverse colon.	Anterior & posterior walls.	Mesocolon. Recurrence at site with metastases in mesentery of small intestine and liver.	Round-cell.	6 months after 1st operation a second revealed inoperable recurrence after first operation.
Meischke, M., 1925	30	F.	Anemia tumor above umbilicus.	.....	Partial gastrectomy.	Posterior wall.	.....	Spindle- cell.	Anemia but no signs of recurrence when last heard of 1 month later.
Straus, Black & Hamburger, 1925 Case I	.....	M.	Emaciation, anemia.	.....	Resection.	Involved 80% stomach wall.	.....	Lymphosarcoma.	Well 4 years later.
Case II	48	F.	Indigestion mass in abdomen.	.....	Resection.	Greater curvature.	Regional lymph nodes.	Lymphsarcoma	Well 4 months later.
Case III	33	.....	Abdominal mass.	.....	Resection.	Diffuse involvement entire wall.	.....	Lymphosarcoma.	Died 5 days later postoperative.
Chand, W., 1926	28	M.	Loss of weight.	6 months.	.....	Cardiac end, greater curvature.	Regional lymph nodes left kidney.	Lymphosarcoma.	Diagnosis established at autopsy.
Farr, C. E., 1926 Case I	34	M.	Epigastric pain.	6 months.	Resection.	Posterior wall lesser curvature.	Regional lymph nodes.	Lymphosarcoma.	Well 3 years later.

Case II	38	M.	Epigastric pain.	1 day.	Resection.	Anterior surface lesser curvature.	Recurrence at site.	Spindle-cell.	Died 1½ years later.
Chandler, I., 1926	38	F.	Mass in abdomen weakness.	6 months.	Resection.	Attached to greater curvature posteriorly by pedicle.	.....	Round-cell.	Well 7 months later. Patient thought she was pregnant and noted growth of tumor for 6 months.
Holmes, Camp & Dresser, 1926	Report 8 cases of sarcoma of stomach seen at Massachusetts General Hospital—No detailed information given. Seven will be counted in this report, as one at autopsy proved not to be primary in stomach.								
Dahs, W., 1926–1927	16	F.	Pain in gastric region.	6 months.	Resection.	Anterior wall.	Regional lymph nodes.	Fibroneurosarcoma.	Condition good at time of publication about 5 years later.
Brander, H., 1927	27	M.	Indigestion.	3 months.	Resection.	Entire gastric wall except cardiac portion.	Regional lymph nodes.	Myxosarcoma.	Died 10 days after operation.
Grek, J., 1927 Case I	24	F.	Tumor in upper abdomen, pain nausea vomiting.	4 months.	"Surgical removal."	"Connected with gastric wall."	.....	Sarcoma.	Fate of patient unknown.
Case II	42	M.	Tumor in skin and abdomen pain, nausea & vomiting.	5–6 weeks.	Removal of skin tumor for diagnosis.	Pyloric region.	Skin and mesentery.	Sarcoma	Autopsy.
Case III	42	M.	Abdominal tumor pain.	6 months.	Operation refused.	.....	.....	Sarcoma.	Autopsy.
Kahan, M., & Seidenburg, A., 1927	23	M.	Epigastric pain.	2 years.	Resection.	Middle and lower 3rds of stomach.	Regional glands and lesser curvature.	Alveolar large round-cell.	This tumor perforated. Death a few hours after operation. Autopsied.
Djorup, F., & Okkels, H., 1927	28	F.	Anemia.	.....	Resection.	Posterior wall greater curvature.			
Bamber, J. M., 1927	19	F.	Vomiting epigastric pain.	18 days.	Resection.	Middle lesser curvature.	Recurrence at site.	Round-cell sarcoma.	Died with recurrence about 2 years later.
Jeffries, J. F., 1927	31	M.	Pain in stomach.	6 months.	Partial gastrectomy.	Pyloric end.	Liver.	Large round-cell.	Two and one-half months later metastases could be palpated in liver. Developments unknown.
O'Callaghan, R. H. L., 1927	54	F.	Indigestion, melena, weight loss.	Few months.	Exploratory with tissue biopsy for diagnosis.	Both curvatures.	None noted.	Sarcoma.	Death 2 weeks after operation.
Cumston, G., 1827	States that he operated on authenticated case, sarcoma of stomach proved microscopically. No details given.								
Bastianelli, P., 1927 Case I	27	M.	Indigestion, tumor at umbilicus.	6 months.	Subtotal resection.	Fundus, antral and pyloric regions.	Recurrence at site.	Small round-cell.	Death seven months later with local recurrence.
Case II	28	F.	Pain & abdominal tumor.	4 months.	Resection.	Greater curvature.	Local reucrrence.	Small round-cell.	Death 6 months late
Case III	30	F.	.....	6 months.	Resection.	From duodenum to cardia.	Local recurrence.	Sarcoma.	Died 9 months later.
Hartman, H., 1927	48	M.	Mass in epigastrium, weakness.	2 months.	Resection.	Posterior wall.	Liver, diaphragm.	Fibromyxosarcoma.	Died 18 days postoperative. This tumor presented in stomach of a case of neurofibromatosis

TABLE I (*Continued*)

Author & Year	Age	Sex	Main Symptoms	Duration	Operation	Location of Tumor	Metastases & Recurrences	Histology of Tumor	Comments
Hartman, H., 1927 (*Continued*)									(von Recklinghausen's disease and presumably arose in a neurofibroma of stomach.
Schlosserer, W., 1928	29	F.	Epigastric tumor and pain.	1 month.	Exploratory laparotomy.	Lesser curvature with infiltration whole stomach.	Regional lymph nodes left kidney, pancreas.	Lymphosarcoma.	Death 8 hours after operation this case gives history of trauma. Was struck in abdomen by chair fracturing 2 lower right ribs 18 months previous to onset of symptoms. Author does not consider this an etiological factor.
Cavina, G., 1928	53	F.	Tumor upper right quadrant.	.....	Gastrorraphy.	Posterior surface greater curvature.	.....	Spindle-cell.	Well at end of 8 months. This tumor was extragastric attached by pedicle lying between stomach transverse colon.
Bullock, H., & Shearman, C., 1928	60	F.	Indigestion.	8 years.	Resection.	Diffuse in gastric wall.	.....	Round-cell.	Condition good 5 months later.
Therstappen, F. J., 1928	.....	.....	Tumor left side abdomen.	.....	Resection.	Exogastric.	.....	Sarcoma of embryonic undifferentiated muscle tissue.	Death 25 days post operative.
Gunsett & Oberling	48	M.	Malnutrition.	.....	Anterior gastro-enterostomy.	Pyloric region.	Regional lymph nodes.	Lymphosarcoma.	Well at end of 5 years.
Freeman, L., 1928	60	M.	Indigestion.	2 years.	Resection.	Diffuse in wall of transverse portion.	Regional lymph nodes.	Lymphosarcoma.	Well at end of 17 months. Received deep x-ray therapy and coleys toxins after operation.
Hall, J. S., 1928	40	M.	Pain after eating.	.....	Resection.	Middle of stomach.	Regional lymph nodes.	Round-cell sarcoma.	Well 4 months later.
Smith, A., 1928	20	M.	Pain & tumor in left abdomen.	5 months.	Exploratory removal of gland for diagnosis.	Involvement whole gastric wall except cardia & pylorus.	Regional lymph nodes.	Sarcoma.	Died 3 weeks post-operative.
Heslop, A., 1928	32	M.	Epigastric pain.	Several weeks.	Resection.	Pedunculated attached lesser curvature.	.....	Sarcoma.	Well at time of report a few months later.
Jaki, J., 1928	23	F.	Tumor in region pylorus, pain.	2 weeks.	Resection.	Anterior wall.	Glands in gastro-colic ligament.	Undifferentiated sarcoma.	Well 6 months later.
Villata, M., 1928	.....	M.	Occasional vomiting & blood stools, loss of	4 years.	Resection of pylorus.	Pyloric portion.	Regional lymph nodes.	Sarcoma originating in reticulo-endothe-	Author is discussing specimen sent to him severe trauma in ab-

			weight.					lial cells.	domen preceded symptoms by 1 month. There was a chronic ulcer, which author thinks followed the trauma and the irridation of this initiated pathological changes resulting in tumor.
Pemberton, J., 1929.....	54	F.	Abdominal mass.	7 years.	Resection & pyloroplasty.	Lesser curvature.	.....	Fibrosarcoma extensive.	Well at time of report a few days after dismissed from hospital.
Curtis, G., & Delaney, P., 1929.....	59	F.	Severe secondary anemia.	.....	Resection & gastroenterostomy.	Pylorus.	.....	Leiomyosarcoma.	In good condition 7½ months later.

on autopsy findings. In the same year, Forni made the most complete and detailed analysis of these tumors which we have been able to find. He brought the total of the reported cases up to 200. In the preceding year and in the same year, we were able to find the report of several cases not included by Forni in his analysis. We have added these cases to those presented by him and have listed the cases reported since that time. The total number of these tumors recorded in the literature through 1929, including those which we now add, is 335. Table 1 presents the salient features of the cases we have reviewed.

FREQUENCY

Sarcomas form only a small percentage of the new growths of the stomach. Various authors, however, have given estimates of their comparative frequency which range from 1 to 8 per cent. Fenwick states that sarcomas occur in 5 to 8 per cent of stomach tumors. Yates made a study of 800 tumors of the stomach and found that 2 per cent of the cases were sarcomas. Ewing estimated their frequency at 1 per cent. Ziesche and Davidsohn in 1909, from the statistics of various German operative clinics and their autopsies, estimated that 1 per cent of the tumors of the stomach are sarcomas. Douglas reported that in a period of eight and a half years (January 1, 1911 to July 1, 1919) there were 702 cases of carcinoma of the stomach diagnosed at Bellevue Hospital, while at the time of his report (1920) the operative records of the Hospital contained only 2 cases of gastric sarcomas. In studying 921 cases which had been diagnosed as gastric cancer, Smithies and Ochsner found 4 sarcomas. Haggard stated that in 27,250 abdominal sections at the Mayo Clinic in five years there were 8 sarcomas of the stomach (Masson) and of 2067 malignancies of the stomach (1908–1920) 13 proved to be sarcomas, or there was 1 sarcoma to 159 carcinomas of the stomach. Brasch called attention to the fact that statistical figures regarding this growth are merely relative, since many cases are assumed to be carcinoma or chronic inflammatory changes associated with chronic gastric ulcer, while subsequent careful histological examination proves them to be sarcomas. He added that Perry and Shaw in going over a series of cases from Guy's Hospital which had been reported as carcinomas, found 4 cases which were really round-celled sarcomas.

The records of the Charity Hospital of New Orleans, from 1906–1929 include 642 diagnoses of sarcoma. Of these sarcomas, 3 were gastric. During this period there were 341 diagnoses of gastric carcinomas. From these records, we may conclude that in this part of the country gastric sarcomas form about ½ per cent of all sarcomas, and about 1 per cent of all malignancies of the stomach.

ETIOLOGY

The specific etiology of sarcomas of the stomach as with all malignant conditions is at this state of our knowledge unknown. We can therefore under this subject, discuss only such factors as have been mentioned as contributing to, or playing a rôle in their development. Such factors are principally trauma, preexisting non-malignant tumors and ulcers, age and sex.

We note a number of cases of gastric sarcoma (Robert, Brooks, Ziesche, Pistocchi, McWhorten, Venturelli, Schlosser, Villata) in which the patients dated the onset of their symptoms to trauma which they suffered. The various authors reporting these cases differ as regards their opinions as to the importance of trauma as an etiological factor. Thus in one case (Pistocchi) a man was perfectly well until he was thrown from a wagon and seriously bruised on the front of the thorax at the height of the last rib. A diagnosis of fracture of the ninth and tenth ribs was made. The pain which developed at the time became increasingly severe and settled in the stomach. Within

two months digestive disturbances appeared, while three months later, pathological changes in the stomach were demonstrated by roentgen ray. The patient died at the end of five months, subsequent autopsy revealing a well developed intragastric, infiltrating lymphosarcoma with metastases to liver, regional lymph nodes and diaphragm. The author remarks that while there is no way of proving that the growth did not exist previous to the trauma, this latter merely serving to awaken it, he is of the opinion that it was probably caused by the trauma. Venturelli reports a case of a woman aged seventy-one who had been in an excellent state of health until she was struck in the left flank with the handle of a broom. Pain, vomiting, loss of appetite, rapidly progressive cachexia ensued, and she died within one month from the date of the trauma. Autopsy revealed a well developed lymphosarcoma of the stomach. The author makes no comment as to the importance of the trauma as an etiological factor. In Villata's case severe abdominal trauma preceded the onset of symptoms by one month. Four years later at autopsy the stomach revealed a chronic ulcer, with a sarcoma originating in the reticuloendothelial cells. The authors consider that the ulcer was initiated by the trauma, and that irritation of this ulcer produced pathological changes resulting in the malignant condition. In Schlosserer's case the patient suffered abdominal trauma eighteen months previous to the onset of symptoms. He does not consider that this was an etiological factor.

Numerous observers report cases in which they feel that benign myomatous growths have taken on malignant properties. Schiller, Kimpton, Fritzsche, Bachrach, and Warner all report cases in which a tumor which was known to have existed for years without causing any symptoms, suddenly began to display malignant features and on removal and histological examination proved to be a sarcoma. Warner states that "just as sarcoma may engraft itself upon a leiomyoma of the uterus, so a like tumor of the stomach may undergo a similar behavior." He furthermore points out that gastric leiomyosarcoma occurs frequently at the same period of life (between thirty and fifty years) that malignant leiomyoma of the uterus develops. Buchrach states that while benign myomata of the gastrointestinal tract are rare, in 1898 the medical literature contained reports of 51 such tumors. He states that in the gastrointestinal tract pure leiomyoma occur, while the corresponding tumors of the uterus are mainly myofibroma, but that in each instance, in an identical manner, the tumors seem to be able to take on malignant properties. Bullock (quoted by Haggard and Fenwick) in writing on benign tumors of the stomach says, "We have not been able to find a single case in the whole literature where a large tumor of the gastric wall was above suspicion of malignancy." In strong contrast to this opinion, is that of Gosset, Bertrand, and Lowey, who upheld the opposite thesis. Of interest in this connection is a case reported by Hartman in which a fibromyosarcoma of the stomach occurred in a case of von Rechlinghausen's disease, the author concluding that it had originated in a neurofibroma. Osler, in discussing von Rechlinghausen's disease, states that the neurofibroma of internal organs may develop sarcomatous changes.

Several writers have questioned the possible origin of a gastric sarcoma from the connective tissue elements of a chronic ulcer. The case of Villata has been previously referred to. Willensky quoted Moser and Kehr to the effect that it is possible for gastric sarcoma to follow ulcer, but says that the relationship is still open to question as it has not been satisfactorily proved. Finsterer reports a case which is interesting in this respect. A man who had suffered gastric disturbances for years was operated on for a duodenal ulcer, at which time a resection of the stomach was done, due to dense adhesions. Two

years later the blindly closed pyloric portion of the stomach was found to contain a polymorphous celled sarcoma which was resected and which caused death by metastases some months later. The value of this instance is lessened, however, by the fact that the specimen from the first operation was not subjected to histological examination. Schindler reports a case in a man of sixty years where death ensued after gastric symptoms of several months' duration. A well developed lymphosarcoma was revealed at autopsy. Between the ages of twenty to thirty this man had suffered with typical signs and symptoms of gastric ulcer with hemorrhage. He received treatment for such a condition and made a recovery. The author concludes that this was a case of sarcoma which developed in a stomach which had been the site of an ulcer. In a case reported by di Giacoma the patient died of advanced pulmonary tuberculosis. At autopsy a round cell sarcoma of the stomach was found, associated with a condition of chronic polypoid gastritis. Stanley Wyard says, referring to gastric sarcoma, "There does not appear to be any connection between them and simple ulcer, though it is possible that irritation may play some part in their production."

While we do not feel that any of the cases which we have cited show conclusive etiological relationship between chronic gastric ulcer and gastric sarcoma, it is our opinion that such a relationship is at least theoretically possible. An ulcer can undoubtedly stimulate prolonged connective tissue formation, and such chronic reactions to irritations have been shown conclusively to act as etiological factors in the production of malignant processes in other regions.

As regards age, the cases reported in the literature have ranged from three and one-half, (Finlayson) to ninety-one years (di Giacoma). Several authors state that the age of greatest incidence is forty to fifty years (Haggard, Garnor and Fairbanks quoted by Frazier). Hertzler, quoted by Warner, says that the disease is so distributed that it cannot be considered a disease of any special age. Scudder says that it is a "disease of the young." Ewing states that the lymphosarcomas occur chiefly in young subjects. Forni estimates that the age of greatest frequency is from forty to sixty years, the average age for the lymphosarcoma being thirty-six years. Chandler in 1926 finds the most common age of occurrence of gastric sarcoma to be 41.6 years, and mentions that according to the last United States census the average age for gastric carcinoma is 61.2 years. Meischke disagrees with Bohmansson's statement that gastric sarcomas "generally occur after forty," and says that most of the cases reported have been in patients under that age. Broders and Mahle in a series of 12 cases of lymphosarcoma found the average age to be 46.25 years, the youngest case being sixteen and the oldest sixty-two. In the series of 135 tumors which we have studied the youngest age was eight years (Demel) while the oldest was eighty-one years (diGiacoma). The average age at onset of symptoms was 36.7 in these cases.

As regards sex, many authors make the statement that there is no special sex predominance in gastric sarcoma (Meischke, Chandler, Douglas). Haggard found that the sexes were equally divided in a series of 66 cases. On the other hand, Broders and Mahle in a series of 12 cases found 11 males. In the 135 cases we reviewed, 73 were males, 43 were females, and in 21 cases we were unable to obtain information regarding sex.

PATHOLOGY

Sarcomas of the stomach are generally grouped accordingly to form into the following types:

Infiltrating, (diffuse, nodular or limited)
Endogastric
Exogastric

Lecene and Petiti accept the following principal types.

1. Diffuse infiltrating form.
2. Nodular form with multiple nodules invading all of the gastric wall.

3. An infiltrating, but limited form.

4. A pedunculated or sessile form over a small portion of the gastric wall.

They add that the first 3 of these forms bear a strong resemblance to carcinoma and are difficult to differentiate grossly. The fourth is a special form of sarcoma and the most frequent of all. We feel that the first classification is a simple workable one, and is the one on which we shall base our discussion.

From a histological point of view, gastric sarcomas are generally said to arise in the submucosa and to then invade the muscular stratum, usually respecting the gastric mucosa and serosa. When the gastric mucosa is affected by this form of malignancy, it is generally due to pressure necrosis and loss of blood supply, rather than to neoplastic invasion.

The tumors may arise from:

1. Connective tissue, true fibrosarcomas.

2. The smooth muscle cells of the wall of the stomach, leiomyosarcoma or malignant leiomyblastoma.

3. The lymphoid nodules, malignant lymphoblastoma, lymphosarcoma or Hodgkin's disease.

4. Endothelial cells of blood vessels or lymph channels, endothelioma, hemangioendothelioblastoma.

In the series of 135 reports we have studied, histological classification of the tumors appeared 100 times, divided as follows:

Lymphosarcomas	39
Round-cell sarcomas	24
Spindle-cell sarcomas	7
Fibrosarcomas	7
Myosarcomas	6
Round and spindle-cell sarcomas	2
Leiomyosarcomas	2
Fibromyxosarcomas	2
Carcinosarcoma	1
Undifferentiated sarcoma	1
Sarcoma originating in reticulo endothelial cells	1
Fibroneurosarcoma	1
Fibromyosarcoma	1
Mixed-cell sarcoma	1
Angiosarcoma	1
Fibrochondrosarcoma	1
Myxosarcoma	1
Polymorphous-cell sarcoma	1

Forni, in his survey, reported the following histological types of growth:

Round-cell sarcoma	98
Spindle-cell sarcoma	72
Particulate-structure sarcoma	39
Polymorphous-cell sarcoma	14

It is to be emphasized as Simon noted in his report that giant-cell and so-called melanosarcomas have not been reported as encountered in the stomach.

As a rule, it may be said that the lymphosarcomas comprise the infiltrating types of growth. They frequently occur in young individuals and offer the worst prognosis of any of the types because, due to their form, they are not so easily extirpated surgically, and are therefore more prone to recur. They also metastasize more readily and at an earlier period in the progress of the tumor. Warner states that this is because of the smallness of the individual cells comprising the tumor, as well as their ameboid properties, thus making it easy for them to enter and diffuse themselves in the various lymphatic spaces. Whether or not cases of Hodgkin's disease should be cited as sarcomas of the stomach depends on whether this disease is considered to be of neoplastic or inflammatory origin. If Hodgkin's disease is of a neoplastic nature it may be considered as a scirrhus lymphosarcoma. A number of authorities adhere to this view. Forman and Scott call attention to the fact that lymphosarcoma of the stomach may be hard to differentiate from other forms of lymphoid overgrowth, especially from glands involved by chronic lymphoid leucemia or aleucemic leucemia, and state that it is only by proper evaluation of clinical data and the relation of the lymphoid overgrowth to the rest of the organism, and the careful study of microscopical sections taken from many portions of the tumor, that a diagnosis of lymphosarcoma can be made. diGiacoma notes that sarcomas of the round-cell type are frequently included in the groups of lymphosarcomas. Warner states in this regard that any round-cell sarcoma bears

the same appearance, and that careful examination with differential stains may prove a specimen which appears to be lymphosarcoma to be either a derivative of smooth muscle fibers or fibroblasts. Rapidly growing and very cellular leiomyosarcomas and fibrosarcomas present small opportunity for complete differentiation of cell types, and may show round cells, not characteristic of their origin. Warner cautions that this fact should be borne in mind, as otherwise one might pronounce the neoplasm a round-celled sarcoma when in reality the growth might be a leiomyosarcoma, fibrosarcoma or lymphosarcoma.

The endogastric and exogastric forms of tumor are more likely to be of histological types other than lymphosarcoma. Cumston states that the exogastric type is the commonest type of sarcoma of the stomach and therefore most interesting to the clinician. These tumors are comparable to other forms of malignancy of benign nature. They metastasize very slowly and are frequently very easily extirpated surgically by cutting off their pedicle. Gosset, Bertrand and Lowey, writing in regard to the pedunculated form of gastric tumors called sarcomas, state that they are in reality clinically and anatomically benign tumors developing at the expense of the neuromuscular apparatus of the stomach, and implantation. Fritzsche, on the contrary, states that thorough histological study of myomatous tumor of the stomach either pedunculated or broad-based would reveal areas of malignancy in the vast majority of cases.

The endogastric type of sarcoma is much less frequent than the infiltrating and exogastric varieties. Bertrand in a series of 70 cases, found only 4. These tumors usually develop beneath the mucosa, pushing it up and at length causing ulceration and sometimes hemorrhage. Cumston states regarding tumors of this type that they are often extremely soft, sometimes even diffluent. When a cross section is made, the cut surfaces look lardaceous, while foci of necrosis and interstitial hemorrhage are detected.

In studying the location of gastric sarcomas it is noted that they do not tend to invade the orifices as do gastric carcinomas. In an analysis of 146 cases, Flebbe found the growth to be located at the pylorus, in 37 cases; greater curvature, 30 cases; posterior wall, 26 cases; lesser curvature, 13 cases; anterior wall, 8 cases; cardia, 3 cases; pylorus and both curvatures, 2 cases; while in 27 cases almost the entire stomach was infiltrated. Cumston says: "From what very little we know, it may be said that sarcoma of the stomach usually respects the gastric orifices which, on the contrary, carcinoma usually invades from the two curvatures and anterior and posterior gastric walls." Brasch states that "while observers differ, the majority agree that the curvatures, especially the greater, are the most frequent sites of origin."

In the cases we surveyed, the location of the tumor was mentioned 100 times as follows:

Greater curvature	17
Pyloric region	16
Lesser curvature	16
Posterior wall	12
Diffuse infiltration	11
Anterior and posterior wall	11
Anterior wall	6
Anterior wall and lesser curvature	3
Posterior wall and lesser curvature	3
Posterior wall and greater curvature	2
Both curvatures	2
Fundus	1

CLINICAL ASPECTS AND DIAGNOSIS

All authors agree that sarcomas of the stomach present no pathognomonic signs or symptoms. The condition is most frequently confused with ulcer or carcinoma of the stomach. There are no cases on record in which a clinical diagnosis of the tumor was established. In Westphalen's case a diagnosis of gastric sarcoma was made by examination of a piece of tissue accidentally obtained during gastric lavage. In Schlesinger's case a correct

diagnosis was established by examination of a portion of a metastatic growth in the rectum. That examination of supposed metastases is not, however, a reliable method for establishment of a diagnosis of a gastric tumor is proved in the case of Leube, where the histological examination of a skin tumor (interpreted to be a metastasis from a gastric tumor) proved to be sarcoma, while later the stomach was revealed to be the site of a carcinoma.

In the cases collected we note that progressive cachexia and anemia, abdominal or epigastric pain, nausea and vomiting, the palpation of an abdominal tumor and hemorrhage (bloody vomitus, or bloody stools) are among the more important symptoms which have served to direct attention to the condition; one or more of them appear in practically every case. These tumors do not tend to given clinical manifestations until late in their course, which accounts for the fact that in numbers of instances the patient is practically moribund before seeking medical advice, and a large inoperable tumor is found at an exploratory laparotomy, or the diagnosis is established at autopsy. We find an impressive proportion of cases in which the original growth in the stomach, although of considerable size, remained "silent" while the metastases produced signs and symptoms which occupied the clinical field. Instances of this sort are seen in the cases of Schlesinger, in which a secondary exudative pleurisy occupied attention, and that of Markiewitz (mentioned by Simon) in which the picture was dominated by large liver metastases which had undergone extensive cystic degeneration. Another instance is that of Cabot's case (15311) reported from the Massachusetts General Hospital, in which the patient's complaint was pain in left chest of four months' duration. Pus was removed from the chest and a diagnosis of subdiaphragmatic abscess was made. At operation an "abscess of spleen" was drained. It was only at autopsy that the true condition was discovered, a lymphosarcoma of the stomach and spleen, the abscess originating in the central portion of the tumor.

The signs and symptoms produced by sarcoma of the stomach are in general those of carcinomas. In most cases of gastric sarcoma which have come to clinical consideration, carcinoma has been the diagnosis rendered. In a series of 12 cases of lymphosarcomas reported by Broders and Mahle, the preoperative diagnosis were carcinoma, in 7 cases; ulcers of the stomach, 1 case; abdominal tumor, (probably inflammatory), 1 case; tumor of the pancreas, 1 case; pyloric obstruction, 1 case. Most authors have stated that clinically, gastric carcinoma and sarcoma cannot be differentiated. There are, however, the following features which are suggestive in attempting a differential diagnosis.

1. Hemorrhage, which though occasionally an outstanding feature (case of Dahs) and at times the direct cause of death (case of Roberts), is less frequent in this condition, and when present is usually a later symptom than in carcinoma. The reason for this is that sarcomas of the stomach are in the vast majority of instances derived from the submucosa and do not invade the mucosa until late.

2. Secretory disturbances and impaired motor functions with resulting symptoms of dyspepsia are less frequent, and are not so likely to be prominent in the case of gastric sarcoma than is the rule with gastric carcinoma In this connection it may be remarked that while some writers report free hydrochloric acid absent, and the presence of lactic acid and the Boas-Oppler bacillus (Schlesinger, Gosset) in sarcomas of the stomach and seem to consider these as suggestive findings, it appears to be the consensus of opinion that they are of no diagnostic or differential value. Monti says that the hydrochloric acid does not disappear until late. Mathiew found hydrochloric acid absent in 11 out of 17 cases. Harlow Brooks found both lactic acid and hydrochloric acid in his

cases, and states that the simultaneous presence of both acids should at least lead to a serious consideration of the possibility of gastric sarcoma. Warner concludes that the absence or presence of free hydrochloric and lactic acid and the presence or absence of occult blood in the stools are very feeble points in the differential diagnosis.

3. Sarcoma does not produce stenosis as frequently, or at as early a stage as does carcinoma, although we do find cases (Klinkert, Schlesinger, Broders and Mahle) in which stenosis was produced and in which the outstanding symptoms were those of pyloric obstruction. Loeper observed 9 cases of stenosis in 120 cases of gastric sarcoma. The explanation for this comoprativer arity of stenosis in gastric sarcamas appears to lie in the fact that carcinomas are more frequently located at the orifices, while sarcomas are more frequently along the curvatures. When stenosis is caused by a sarcoma it is more likely to be due to a massive infiltration of the stomach wall, rather than a narrowing of the pyloric orifice. Massive infiltration may lead to a dilatation of the stomach wall and gaping of the pylorus.

4. *X*-ray examination will usually furnish evidences of a gastric neoplasm but does not give information enabling a positive diagnosis of sarcoma. Douglas states that while the *x*-ray is the greatest aid in making a diagnosis of the presence of an operative lesion, "a positive diagnosis of sarcoma could not possibly be made by radiographic examination." Strauss, Black and Friedman and Hamburger state that due to the fact that the sarcomas infiltrate the muscularis and submucosa, it is many times impossible to demonstrate it by means of the fluoroscope or *x*-ray. Cutler and Smith state that "the *x*-ray alone gives a clue to the differential diagnosis since the sarcoma projects into the lumen rather than produces an ulcer, with the typical filling defect." Therstappen on the contrary, says that the roentgen picture cannot be said to be really reliable or characteristic in sarcoma of the stomach.

5. Schindler believes that if one is familiar with the picture the diagnosis of diffuse lymphosarcoma of the stomach is possible with the gastroscope. The author states that in his case he did not make a correct diagnosis because he had never seen such a case before, but now he realizes that the condition "may present a characteristic picture in the gastroscope, which is entirely different from all other diseases of the stomach."

In spite of all these various views regarding differential diagnosis or even a diagnosis of sarcoma of the stomach, we have as previously noted, found no cases of a correct diagnosis being established by means other than an exploratory laparotomy or autopsy except in the previously mentioned case of Westphalen. In Schlesinger's case, which at subsequent operation proved to be a gastric sarcoma, the following preoperative diagnosis was made: "Pyloric obstruction, probably syphilis or sarcoma. Carcinoma not probable."

PROGNOSIS

In view of the malignant nature of the disease, prognosis is necessarily grave. Ruffin states that in his search of the literature the only report of a cure is that of Ruppert. The case was that of an old woman of seventy-eight on whom had been done a nearly complete gastrectomy fourteen years previously for a large lymphosarcoma which occupied almost the whole length of the stomach with involvement of the glands of the greater and lesser omentum and the region of the cardia. Finsterer reports a case of lympho-sarcoma in a man twenty years of age which he operated nine years previously and who at the time of the report was in excellent health. Koettlitz states that while the condition is hopeless without treatment, due to the relative rareness of metastases, and the fact that some sarcomas are pedunculated, operation offers a certain chance of success. He then quotes Gosset's

statistics in which 39 cases of extragastric sarcoma showed 19 survivals after operation, while in 22 cases of the intragastric type, 8 recovered after resection. Warner considers that due to the fact that metastases are, as a rule, of later occurrence in the case of sarcoma of the stomach than of carcinoma of the same location, surgery is more promising in the former condition. He states that in certain instances where a tumor, if carcinoma, would hardly be worth the trial of removal, it might be attacked with some of success, if sarcoma, because of the improbability of metastases and lesser degree of malignancy. Scudder likewise shares this opinion and states that in the absence of metastases, a radical operation for sarcoma of the stomach appears to hold out better chances of cure than with a carcinoma of the same organ. Noordenbos makes the following statement: "The prospects of operation for gastric sarcoma are worse than in cancer of the stomach, the operative mortality being even larger, but when the patients have once tolerated their operation their prospects of survival are better. In absence of metastases the prognosis of radical operation is better in sarcoma, than in cancer of this organ."

The type of the sarcomatous growth is a consideration in offering a prognosis. Thus Brasch states that the average duration of untreated round cell types, (including lymphosarcoma), is about fifteen months, that of spindle-celled types twenty-four to thirty-two months and that of myosarcomas, three and one half years. Both Brasch and Warner state that the lymphosarcoma and round cell sarcomas are more apt to produce metastases and at an earlier date in the progress of the neoplasm than are any other non-epithelial tumors of the stomach and are also more apt to recur after removal. Warner attributes these facts as already noted to the small size of the cells and their ameboid properties. Both authors conclude that even the most prolific varieties, the lymphosarcomata and the leiomyosarcomata, show less tendency to recur after excision than do carcinoma of the same region and for these reasons offer a better prognosis. Brasch states that this view is substantiated by the fact that the average duration of life after the onset of symptoms is longer than in cases of carcinoma, being frequently as much as three to four years. Kuchebski states that gastric sarcoma may be considered as less malignant than gastric carcinoma because of the fact that it metastasizes more rarely. He concludes, however, that this alters the prognosis very little as the patient dies as a rule in one or two years, just as in the case of carcinoma. He says that the only hope lies in a radical operation to guard against metastases and recurrences and only poor results can be expected from a gastroenterostomy, as the infiltrated walls do not easily anastomose.

The prognosis in a case of gastric sarcoma is undoubtedly influenced by the histological type and the form. All authorities agree that the pedunculated growth offers a much better prognosis than does the infiltrating variety. The apparent explanation for this is that a pedunculated growth offers a better chance for surgical extirpation and furthermore that they are most frequently of the spindle-cell type which is a relatively slow type of growth, tending to remain circumscribed and not readily metastasizing. On the contrary the diffuse form, which is almost always lymphosarcoma or round cell sarcoma, offers a worse prognosis than does carcinoma. Cavina says: "The prognosis does not seem to be very serious in extragastric spindle-cell sarcoma." His patient at the time of the report was well eight months after operation for removal of such a tumor. On the other hand, Gunsett and Oberling report the persistence of cure at the end of five years in a case of far advanced lymphosarcoma. It was considered inoperable, and an anterior entero-enterostomy was done followed by several courses of *x*-ray therapy. They consider that these tumors are extremely radio-

sensitive and that such therapy will favor the prognosis.

Klinkert concludes that since the more malignant varieties, the round cell and lymphosarcomas, are comparatively more frequent in young individuals than the fibrosarcomas and myosarcomas which occur in later years, it would seem that the prognosis is worse in youth.

Haggard gives the following results in a series of 46 cases.

1. Died	20 cases
2. Recurrence	3 cases
3. Recovered and well for varying periods of from four to ten years	41 cases

In considering the prognosis, we may mention that gastric carcinomata have a greater tendency to perforate than have gastric carcinomata. Kolbe reports an interesting case of fibrosarcoma which developed silently until it perforated through adhesions into the abdominal wall, producing an abscess. About six months later this abscess gave rise to an acute peritonitis which caused death, and the diagnosis was established at autopsy.

Demel and Fritzsche also report cases which perforated. Gastric sarcoma have been said to perforate in 8 per cent of cases (Hesse quoted by Douglas). We would not be inclined from our studies to put the percentage nearly as high. Klaus writes regarding gastric sarcoma, "The prognosis of lymphosarcoma is bad. That of myosarcoma and fibrosarcoma offers a more favorable prognosis than does carcinoma."

SUMMARY AND CONCLUSIONS

1. Sarcoma of the stomach is a relatively rare form of neoplasm being most generally estimated as forming about 1 per cent of gastric malignancies and about 0.25 per cent of sarcomas in general.

2. They present no pathognomonic features which would allow them to be diagnosed by clinical or laboratory tests.

3. Diagnosis is established only by histological examination of an excised portion of the tumor or of one of its metastases.

4. Clinically and by roentgen ray, gastric sarcomas are most frequently diagnosed as carcinoma or chronic ulcer, and it does not appear possible to make a differential diagnosis before operation.

5. While age is not of importance in the differential diagnosis, gastric sarcoma tends to occur in younger individuals than does gastric carcinoma, the average age for the onset of symptoms being 41.6 years for the former and 61.2 years for the latter. The average occurrence age for gastric lymphosarcoma is even lower than that of gastric sarcoma in general, being thirty-six years.

6. While the prognosis of gastric sarcoma is poor, it is frequently said to be better than that of carcinoma of the same region, due to the fact that it does not seem to metastasize so readily or at such an early stage in its development.

7. The essential etiology of these tumors is naturally unknown. Trauma, preexisting neoplasms of a benign nature and chronic ulcers have been discussed as possible etiological factors.

8. Lymphosarcomas of the stomach are usually of the infiltrating variety, occur in younger individuals than do the spindle-celled type of tumors which are more frequently pedunculated. These latter offer a much better prognosis than do the former due to the fact that they metastasize more slowly and are more readily extirpated surgically.

9. Three hundred and thirty-five cases of sarcoma of the stomach, including the four herein reported, are recorded in medical literature through the year 1929. [For Bibliography see Author's reprints.]

EVALUATION OF THE X-RAY EXAMINATION IN DUODENAL ULCER*

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EVEN the radiological consideration of duodenal ulcer should take into account the fact that this condition is a disease and not a lesion. The etiology is evidently not at all clearly understood. The focal infection theory sponsored by Rosenow has many adherents but the evidence for it does not seem at all conclusive to perhaps the majority of clinicians. It has been impossible to produce at all uniformly in laboratory animals anything closely resembling the typical peptic ulcer which occurs in man either by inoculation of specific strains of bacteria, by traumatism, by maintenance of increased acidity or by any other means. It was Virchow's idea that ulcer was caused by thrombi or emboli and this theory still has supporters among pathologists. Others consider the initial lesion to lie in the autonomic nervous system and link the condition with the supposed functional disturbance known as vagotonia, which is presumed by this school to precede the actual breaking down of the mucous membrane resulting in ulcer. Heller modifies this view by assuming the occurrence of toxic or infectious injury to the vagus, causing spasm of the intrinsic musculature followed by ulceration.

Local vasoneurotic disturbances involving the so-called ulcer bearing area and resulting in a spotted hyperemia of the mucosa with dilatation of the capillaries apparently accompanies many peptic ulcers and is to a rather definite degree characteristic of the condition which precedes ulcer or makes it possible.

The failure after many years of intensive investigation to discover any constant, etiological factor, plus the well-known tendency of ulcer to recur after prolonged absence of symptoms and after apparent medical or surgical cure, leads most clinicians of long experience to the belief that there is an underlying ulcer diathesis, the nature of which is as yet quite unknown. Almost universally the presence of hyperacidity has been considered not only a diagnostic point of great value but the chief factor in preventing the ulcer from healing. Yet even Sippy, the strongest and most logical advocate of this theory, admitted that in every series of ulcer cases there was a certain fairly large number of normal acidities and a few subacidities or even complete achlorhydrias. It is very difficult to explain the persistence of ulcer in these cases, if one adheres to the acid theory, and the occurrence of normal or subacidities in, according to some authors, as high as 30 per cent of ulcer cases makes gastric analysis of much less value from the diagnostic standpoint. Its importance is further lessened by the fact that there are a few, persistent instances of hyperchlorhydria which fail to develop ulcer over even a rather long period.

The presence of occult or visible blood in the stomach contents after aspiration, or in the stool after the patient has been on a meat free diet for several days is of some value in the diagnosis of ulcer, provided a number of confusing factors can be excluded. This finding is, for instance, quite the rule in malignancy of the stomach; apparently it occurs occasionally from traumatism at the time of intubation, and certainly it frequently accompanies carcinoma of the biliary tract. It is, of course, of value only if positive, since not all ulcers bleed and those which do, do so only occasionally. It seems evident, then, that the chemical and microscopic investigation of stomach contents and stool, while still undoubtedly deserving an important place in the routine

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of diagnosis, are not in themselves dependable or sufficient.

The value of history has been stressed by all clinicians and perhaps especially by Sippy, and cannot be denied. Provided the clinician is sufficiently familiar with the symptoms of the disorder, he can in the typical case at any stage hazard a fairly accurate conclusion as to the location and character of the lesion and the extent of complications, if any, from the history alone. The clinician who knows ulcer well enough to do this, however, will perhaps be the first to agree that other conditions presenting no organic lesion in the stomach or duodenum can, under exceptional circumstances, simulate very closely and to a very confusing extent the picture of duodenal ulcer.

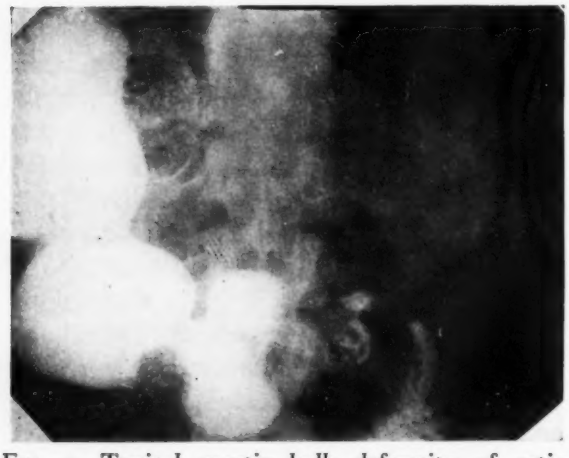

Fig. 1. Typical spastic bulb deformity of active duodenal ulcer with marked hyperperistalsis. Failure to outline pylorus completely due to posture and not to disease.

It is apparently the rule that duodenal ulcer does not come under exhaustive clinical investigation until several years after the inception of the symptoms and even if they were perfectly typical during the first few months of the disease, they are quite apt to diverge widely from the textbook picture in later years. Perhaps everyone who has seen a considerable number of ulcer cases will recall instances in which the symptom complained of most bitterly was a pain in the upper lumbar or lower dorsal region somewhat related to food-taking in its times of maximum severity but by no means as definitely so as the ordinary type of chemical discomfort. I have on two or three occasions been called upon to examine the spine in an effort to trace the cause of this type of pain when it proved later to have been due to duodenal ulcer. Oddly enough, the back pains are temporarily relieved by alkalies just as is the usual epigastric discomfort.

Fig. 2. Typical marked bulb deformity from ulcer. c indicates crater.

Ulcers are apt to be accompanied by a greater or less degree of spastic disturbance of the colon which probably accounts for the fact that in many instances the most annoying symptoms in established duodenal ulcer are referable to the colon rather than to the stomach or duodenum. Distressing accumulation of intestinal gas is very frequently part of this picture and alternating periods of constipation and diarrhea quite apart from effects of diet and medication are sometimes more conspicuous, so far as the patient's voluntary comment is concerned, than the chemical discomfort. Occasional periods of persist-

ent soreness and tenderness in the epigastrium lasting for days or perhaps for weeks and not relieved by food or alkalies are

Fig. 3. Typical bulb deformity from ulcer. c indicates crater. Note spastic incisura on both borders just above and left of crater.

not at all uncommon in established ulcer and are also, of course, the usual preliminaries to slow perforation. These attacks of soreness are presumably traceable to perigastritis or periduodenitis but they may recur from time to time for years without evidence of extensive inflammatory reaction about the pylorus or duodenum being visible at operation and without being followed by perforation.

Duodenitis is probably very frequently a cause of atypical symptoms and apparently there is occasionally a duodenitis with a rather characteristic intolerance of the duodenum to barium which often precedes the actual establishment of ulcer or at least which precedes any deformity which can be visualized. It has recently been pointed out that the institution of the ulcer regimen at this stage, rigidly adhered to for three or four weeks, may possibly prevent the formation of ulcer.

It is evident then that it is not always possible to diagnose duodenal ulcer either from the history or from the laboratory findings and since not every case which presents a fairly typical ulcer syndrome is a case of duodenal ulcer, how can the condition be recognized with any satis-

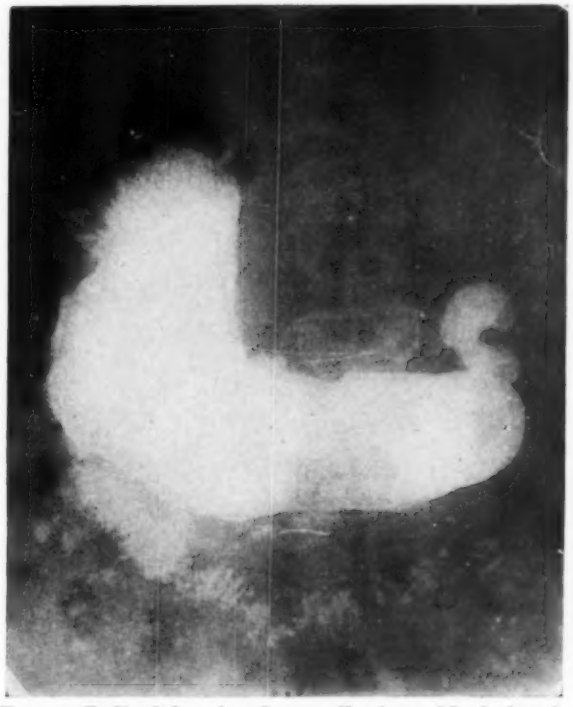

Fig. 4. Bulb deformity from adhesions. No lesion in duodenum.

factory degree of certainty? The answer is the same as would be given with regard to an ulcer on the leg: by looking at it. There is, practically speaking, no other lesion than duodenal ulcer which will produce a typical deformity of the duodenal bulb and there is no practical possibility of an ulcerated duodenal bulb presenting a normal *x*-ray appearance. Therefore, the *x*-ray diagnosis of duodenal ulcershould be and is, in the hands of competent and experienced gastrointestinal radiologists, as nearly accurate as any diagnostic measure.

The first part of the duodenum, referred to by radiologists as the cap or bulb, is the first inch or inch and a half of the duodenum distal to the pyloric ring and it acquires these appellations from its shape, as visualized when completely filled with barium, under the fluoroscopic screen. It is the site of practically all duodenal ulcers. It is almost never the

site of malignancy and it is with extreme rarity that any other intrinsic lesion except ulcer is found there. The bulb may be visualized with greater or less difficulty according to the habitus of the individual and according to the rapidity with which it fills and empties. Given, however, a sufficienct amount of patience and ingenuity, it can be visualized in a filled state on the fluoroscopic screen almost without exception in all individuals and can, as a rule, though not always, satisfactorily be shown on films.

FIG. 5. Marked dilatation of first part of duodenum due to ulcer at distal end of bulb with obstruction at that point.

It remains then, so far as the diagnosis of this particular lesion is concerned, for the radiologist to visualize the bulb in a filled state for a sufficient long period and in a sufficient number of projections to satisfy himself that it really is, or is not, of normal contour.

If it is normal in outline (and the normal outline varies somewhat with the shape of the stomach) certainly it is not ulcerated. If it is deformed, the deformity must be due either to ulcer with spasm or to adhesions; and the latter type of deformity can, as a rule, be readily distinguished from that of the ulcer.

Oftentimes the most conspicuous part of the deformity is that due to spasm and this is, to a considerable degree, a measure of the activity of the ulcer.

FIG. 6. Small, contracted, deformed bulb with early obstruction.

Thus a healed ulcer, if of any size, is very apt to cause a persistent deformity of the bulb throughout the life of the individual, but is no longer accompanied by spasm. It should then be possible to affirm not only the presence of ulcer but its size, its location and the extent to which it is accompanied by spasm. These data are of equal importance with the demonstration of its existence, in forming a basis for prognosis or treatment.

It occasionally happens that a competent radiologist makes a diagnosis of duodenal ulcer on such excellent grounds that he considers it unassailable and yet, when the surgeon opens the abdomen shortly after, he is unable to find any evidence of the lesion. These occurrences are very embarrassing to all concerned and naturally the blame is ultimately passed to the radiologist where, indeed it would seem, on the face of the matter

to belong. When Carman at the Mayo Clinic was engaged several years ago in establishing a direct diagnosis of duodenal ulcer by visualization of the lesion, this sequence occurred several times and for some months he accepted the burden of these apparent errors with decreasing cheerfulness and with perhaps some growing doubts as to the accuracy of his own conclusions. Then he began to insist on an investigation of the inside of the duodenum instead of accepting the surgeon's negative conclusions based on lack of characteristic stippling of the peritoneum and lack of induration, and this led to the true explanation, namely: that there existed in these instances very superficial mucous ulcers which were, however, surrounded by an inflammatory zone of sufficient extent to cause the characteristic spasm. These cases were true duodenal ulcers but they were, histologically speaking, very early and had not yet produced the characteristic external changes. Since the spasm which is so prominent to the vision of the radiologist is entirely relaxed by the anesthesia, these duodenal bulbs looked perfectly normal and since no induration was present, they felt normal: yet the ulcer was there and visible enough when the mucosa was exposed. I have had the experience of having this type of lesion chalked up against me as an error in diagnosis only to reexamine the patient a few months later and find a well-established, typical ulcer which at a second operation was equally evident to the surgeon.

A case which recently came under observation is decidedly in point.

The patient, a physician himself interested in gastroenterology, had had for approximately ten years very definite and typical symptoms of duodenal ulcer relieved from time to time by the ordinary routine medical treatment. After several months of relative freedom from symptoms a massive hemorrhage suddenly occurred and as it could not be controlled by the usual medical measures, surgical interference was resorted to. Approximately four years before operation, this physician had been examined by one of the most prominent radiologists in the country and a definite

Fig. 7. Contracted, deformed bulb with fairly high-grade obstruction.

and typical duodenal ulcer had been located on the lesser curvature surface of the bulb near the base. In view of this fact the patient explained to the surgeon that the bleeding lesion would probably be found at this point and would undoubtedly be an old, calloused ulcer of considerable extent. When the duodenum was examined through the incision the surgeon at first had the impression that no ulcer was present. There was no scar. There was no callus or thickening of the wall. There was not even any stippling of the peritoneal surface. The duodenum was then opened and with some difficulty the bleeding lesion was located. It was found to be a very shallow, superficial affair at the distal end of the bulb. There was not even any infiltration of the wall and in fact everything in its appearance pointed to its being an extremely recent affair. The only possible conclusion was that the ulcer formerly observed and presumably accountable for years of symptoms had healed leaving no palpable scar and that the hemorrhagic lesion was a recent and separate one.

This case brings out in an unusually clear way several points of considerable interest. It supports the hypothesis of an underlying predisposition to ulcer showing

that the original lesion may heal even without scar formation and another form at some later date. It may offer an explanation of the often-quoted, somewhat cynical remark that after an ulcer has been cured a certain number of times by medical measures it should be subjected to surgical intervention. Probably ulcers are often actually cured by medical handling and the recurrence of symptoms is due not to a reactivation of the old ulcer, but to the formation of a new one. Hemorrhage from a duodenal ulcer is, of course, to be classed as an accident more than as a symptom of the disease. In other words, it is entirely dependent on the location of the ulcer and the accident of its eroding a vessel, large or small, an occurrence which may not take place with an extremely extensive, old ulcer but which, on the other hand, can and often does occur with a very recent and very superficial lesion. Examination of patients with an undoubted clinical history of hemorrhage from a duodenal ulcer within a reasonably short time after the occurrence of the hemorrhage often fails to disclose any extensive or characteristic deformity.

A diagnosis is often made by the presence of the deformity produced by spasm when the lesion itself is very small and may be difficult or impossible to visualize. Spasm in the first part of the duodenum is believed to be caused by reflex irritation from a hyperemic inflammatory zone about the ulcer through a short arc intrinsic in the duodenum itself. Hence, it is not relaxed by the administration of even large doses of belladonna while, on the other hand, it is completely relaxed by general anesthesia.

For this reason, antispasmodics are of little value in the re-examination of suspected duodenal ulcer cases in which the first examination has been for some reason unsatisfactory or inconclusive. They are of considerable value in the reexamination of cases of suspected gastric ulcer.

It was formerly the rule that approximately one-third of the cases diagnosed by *x*-ray were identified only by the so-called indirect signs consisting of enlarged stomach, hypertonus and hyperperistalsis and a considerable four and a half hour or six hour residue coupled with inability to visualize a normal duodenal bulb. In more recent years this has been considered a somewhat unsatisfactory type of diagnosis and the feeling has obtained that with sufficient care the bulb or what remains of it should be capable of visualization.

The amount of residue present in the stomach after the normal emptying period has passed may vary considerably from time to time even in the presence of rather high-grade organic obstruction. Thus it is not infrequent to see a patient with a 50 per cent four and a half hour residue progress to a normal emptying time after a few weeks on a carefully supervised ulcer regimen. This does not, of course, mean that the diet (and the alkalies, if these are given) have brought about the absorption of any organic obstruction due to scar tissue but rather that most of the obstruction was due to an inflammatory process which has subsided under the measures adopted. These are probably early cases of true organic obstruction which will, in the absence of surgical relief, go on to high-grade stenosis.

It is essential for the radiologist to bear in mind certain rare developments in a course of duodenal ulceration which occasionally produce confusing and atypical *x*-ray appearances. One of these, illustrated in Figure 5 is stenosis of the duodenum at the junction of the first and second portions, just caudad to the bulb with consequent dilatation or sacculation of the bulb. This is to be distinguished from duodenal diverticulum which it might suggest. It should be recalled that through subacute perforation a spontaneous anastomosis may form between the bulb and the distal duodenum. This is evidently an extremely rare occurrence and has never come under the writer's observation although the comparable instance of spon-

taneous anastomosis between the ulcerated stomach and the duodenum, jejunum or colon is observed from time to time with equal potentialities for confusing the diagnosis unless these possibilities be kept in mind.

Finally, the radiologist is not infrequently called upon to reexamine known cases of duodenal ulceration after treatment or operation with a view to determining the activity of the lesion. Such examinations are probably considerably less satisfactory in the case of duodenal ulcers than in gastric ulceration. If a definite ulcer crater has been observed prior to operation or treatment and cannot be demonstrated on reexamination, it is perhaps reasonably safe to assume that the ulcer has healed. One must not expect after definite ulceration has once occurred, whether or not there has been operative interference, to observe a smooth bulb of normal contour. Some conclusions may be drawn from the presence or absence of spasm. Delayed motility, if present, can, of course, be determined. Reports of such examinations should, however, be somewhat guarded.

CARCINOID TUMORS OF THE SMALL INTESTINES*

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CANCER is notably infrequent in the small intestine. From 0.1 to 0.5 per cent of all malignant tumors are situated in this region. This infrequency is singular when we consider the relatively large surface area of intestinal mucosa which is exposed to possible irritation. Carcinoma of the small intestine occurs in subjects of comparatively early life; the average age is usually about forty years although many cases occur in the decade of twenty to thirty. From three-fifths to two-thirds of all the subjects are males. The common histological type of cancer in this location is the ordinary adenocarcinoma. The carcinoid tumors are rare and constitute an interesting clinical and pathological study.

Carcinoid tumors are quite frequent in the appendix, particularly in instances of so-called obliterative appendicitis. The ileum is next in frequency as the site of their occurrence; other infrequent locations are the duodenum, jejunum, colon, rectum and Meckel's diverticulum (Stewart and Taylor). These tumors are frequently multicentric in origin. They are button-like, circumscribed but not encapsulated tumors invariably situated on the anti-mesenteric side of the bowel. They may lead to a slight degree of stenosis and occasionally are the cause of intussusception. They may exist for a long time without ulceration. The tumor cuts without grating; the cut surface is waxy, firm and yellowish white.

THEORIES OF HISTOGENESIS

We shall give briefly the various theories concerning the histogenesis of this peculiar tumor, naming these theories in the chronological order of their publication.

(a) *Lubarsch* in 1888 employed serial sectioning of these tumors and found that certain cells in the apices of Lieberkuehn's crypts broke through the basement membranes and formed independent cell groups. Lubarsch observed the growth of these cell masses into lymphatic vessels and labeled this primary tumor "carcinoma cylindromatosum." He differentiated these tumors from other adenocarcinomas occurring in the alimentary tract by certain peculiar features, namely, multicentric origin, absence of true glandular structure, disinclination to metastasize and cellular dissimilarity.

(b) *Krompecher* in 1903 designated this tumor as a "basilome" and declared its origin to be from the cylindrical cells of the intestine. Later in 1919 Krompecher modified his theory and asserted that these tumors were identical with the basal cell carcinomas of the skin. He observed three types, solid, adenoid and cylindromatous. At this time he considered two possible origins:

1. From the cylindrical cells of the intestine.

2. From the basal cells of the crypts of Lieberkuehn.

(c) *Schmidt* in 1905 described certain basal cells occurring in the intestinal mucosa which he considered to be the same as the cells described by Kultchitzky in 1897.

(d) *Oberndorfer* in 1907 reported six instances of these tumors and first proposed the name "carcinoid."

(e) *Trappe* in 1907 expounded the

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theory that carcinoid tumors and adenomyomas of the small intestine had a common origin from pancreatic rests. The

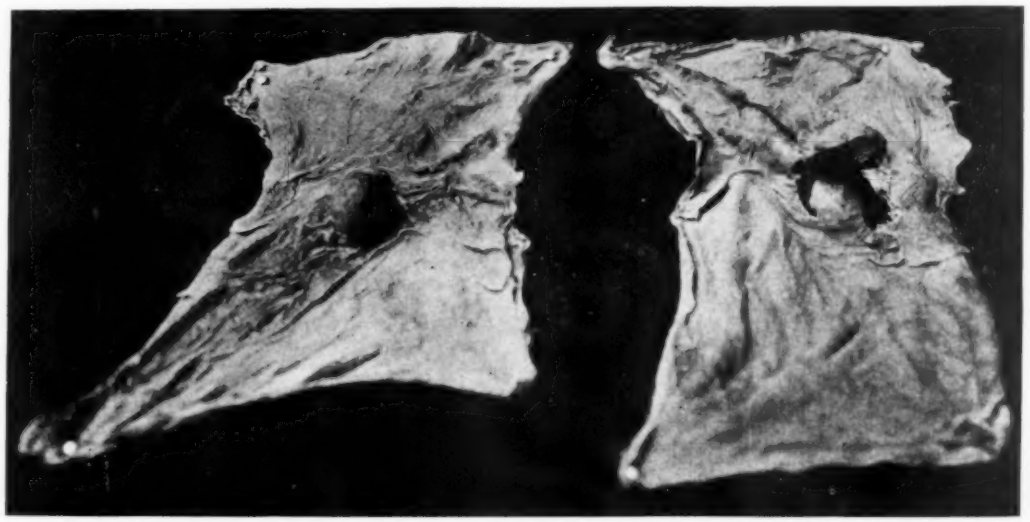

Fig. 1. Multiple carcinoid tumors of ileum. Mucosal surface uppermost to demonstrate gross characteristics of these neoplasms.

carcinoid tumor was less differentiated than the adenomyomas and therefore was assumed to have a greater capacity for growth.

(f) *Burkbard* in 1909 agreed with Krompecher on the similarity of these tumors with basal cell cancers of the skin. His several reasons for this assumption were based on certain features of the carcinoid tumor, namely, its failure to differentiate, its low degree of malignancy, its tendency not to metastasize and its seeming continuity with the intestinal epithelium. He decried the possibility of an origin from pancreatic rests because of his inability to detect ducts or pancreatic acini in the tumor.

(g) *Toenniessen* in 1910 considered these tumors as originating in submucous rests possibly of Langerhans' islands. He suggested a relationship to the endocrine system.

(h) *Huebschmann* in 1910 also suggested the possibility that these carcinoid tumors might evolve from the Schmidt chromaffin cells lying in Lieberkuehn's crypts.

(i) *Masson* in 1910 made a thorough study of these peculiar cells and observed lipoids, vacuoles and silver-positive granules in the cytoplasm. On the basis of this observation he emphasized the resemblance of these cells to the medullary cells of the adrenal gland and to other cells of the chromaffin endocrine system. In 1912 and later he elaborated his theory to prove conclusively for the first time the exact nature and origin of these carcinoid tumors. We will quote this theory in detail.

> Certain intestinal cells (of Kulchitzky) bud out and migrate into the nerves. Here they become argentaffine. They differentiate into various forms, cylindrical cells grouped in rosettes or vesicles, cells of ganglion type, of Schwannian type, neurocrine cells. The nerves containing them grow and become neuromata, or disappear if the cells themselves disappear. Carcinoids result from the autonomous proliferation of the isolated neurocrine cells present in the neuromata . . . Either the nerve plexus is of sympathetic origin and proliferates under the irritating or secretory influence of the cells which have emigrated from the intestinal epithelium . . . or the periglandular plexus consists of a mixture of fibers, some sympathetic, others belonging to another nervous system, autonomous and autochthonous, of entodermic origin.

In conclusion, Masson considers the possibility of a neuro-entodermic placode

of which the cells of Kulchitzky might be the sole manifestation in normal conditions.

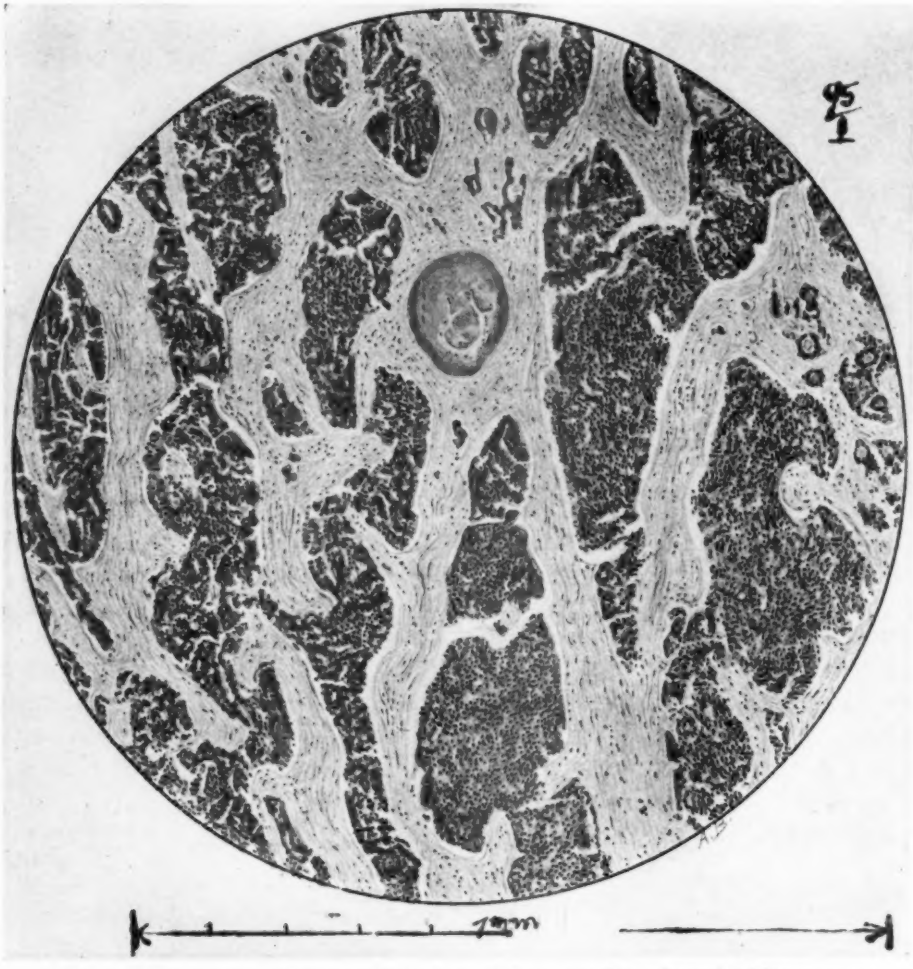

Fig. 2. Low power microscopical view of deep portion of carcinoid tumor.

(j) *Luce* in 1912 termed these tumors basal cell carcinomas of the mucous membrane.

(k) *Saltykow* in 1912 studied 7 cases and came to the conclusion that these tumors originated from pancreatic rests of Langerhans' islands. He defended his argument by calling attention to the fact that it was possible for the Langerhans cells to be included in the intestine because they differentiate earlier in the embryo than does intestinal muscle.

(l) *Hasegawa* in 1923 agreed in general with Masson's theory as to the origin of these tumors from Schmidt's cells or the cells of Kulchitzky.

(m) *Aschoff* in 1923 called these tumors "Schleimhaut" nevi, or mucous membrane nevi.

(n) *Engel* in 1923 labeled these tumors "choristoblastomas" and admitted their origin in embryonic rests.

An analysis of the numerous theories of histogenesis given here permits us to classify them in three main groups:

1. A type of basal cell cancer (Krompecker, Burkhard) or nevus (Aschoff) of mucous membrane.

2. A tumor originating in embryonal rests particularly of pancreatic islands. This hypothesis corresponds to the Cohnheim-Durante theory of tumor origin and is accepted by Saltykow, Engel, Trappe and Toeniessen.

3. A tumor originating from the cells of the intestinal epithelium: Lubarsch, Hueb-

schmann, Masson and Hasegawa. Masson's conception of nerve relationship is the most logical explanation to-date.

size, rich in chromatin, placed in the center of the cells, having a capacity to stain deeply and containing prominent

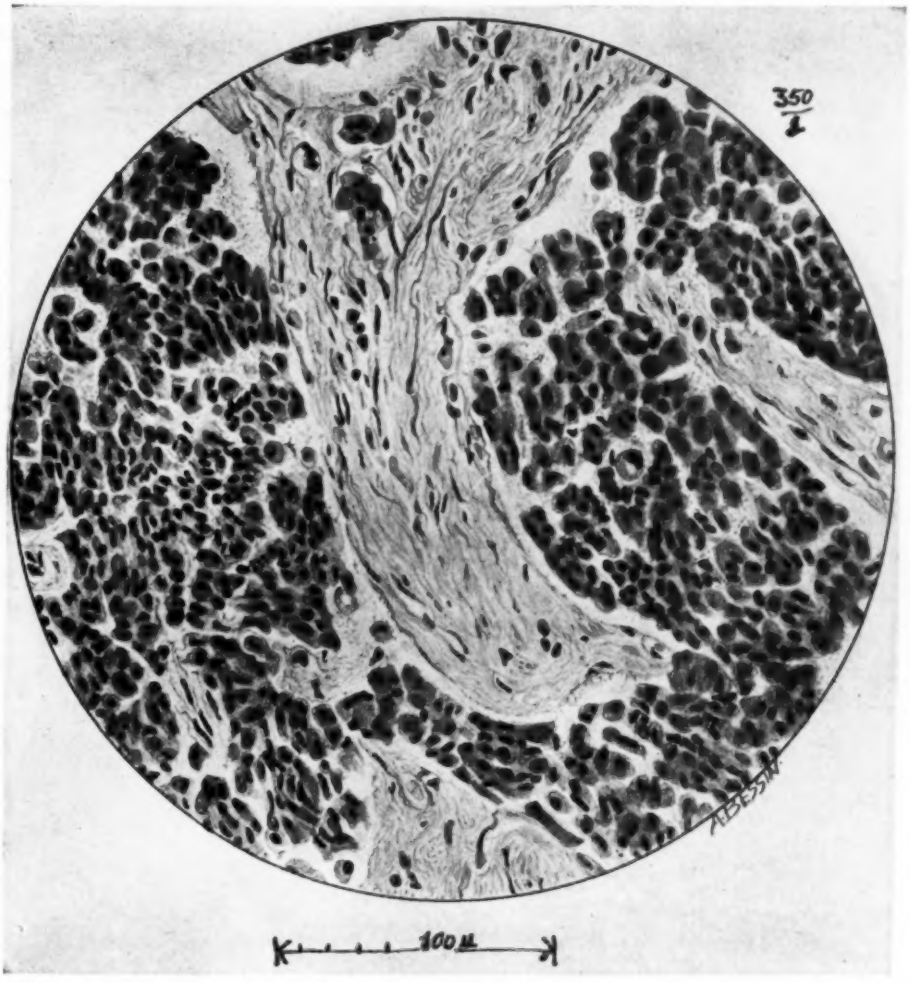

Fig. 3. High power microscopical view of same field as in Fig. 2. To show solid cords of undifferentiated cells and character of nuclei. Definite morphological resemblance to basal cell carcinoma.

MICROSCOPICAL APPEARANCE

The tumor cells are grouped in nests composed of solid cords in some of which a pseudoglandular arrangement is seen. This gland-like arrangement is more common in the benign carcinoid tumors and is found in the superficial portion of the tumor near the intestinal mucosa, rather than in the depth of the intestine where the cords of cells are usually solid. There are no true alveoli. The major portion of the tumor is composed of undifferentiated cell masses. The nuclei are large, oval to round and regular in shape, uniform in nucleoli. The cytoplasm stains indifferently, is moderate in amount and occasionally contains reddish brown granules when stained by the ordinary methods.

Hasegawa demonstrated intracellular lipoids in these cells on frozen section, a finding said to distinguish these tumor cells from the cells of true adenocarcinoma.

There are certain granules in the cytoplasm of these cells which have an affinity for silver. These so-called argentaffine granules can be shown by proper staining methods in the Schmidt or Kulchitzky cells *in situ* in the crypts of Lieberkuehn

as well as in the carcinoid tumors. In fact, this staining reaction has been of great importance in studying the histogenesis of these carcinoid tumors. When the tumor undergoes malignant degeneration it loses a proportionately large amount of these argentaffine granules. Therefore some of the malignant carcinoid tumors do not react positively to silver stains. For instance, in 2 of Hasegawa's cases in which malignant changes were associated with the carcinoid tumor, no argentaffine cells could be found. The same condition obtained in the tumor reported herein.

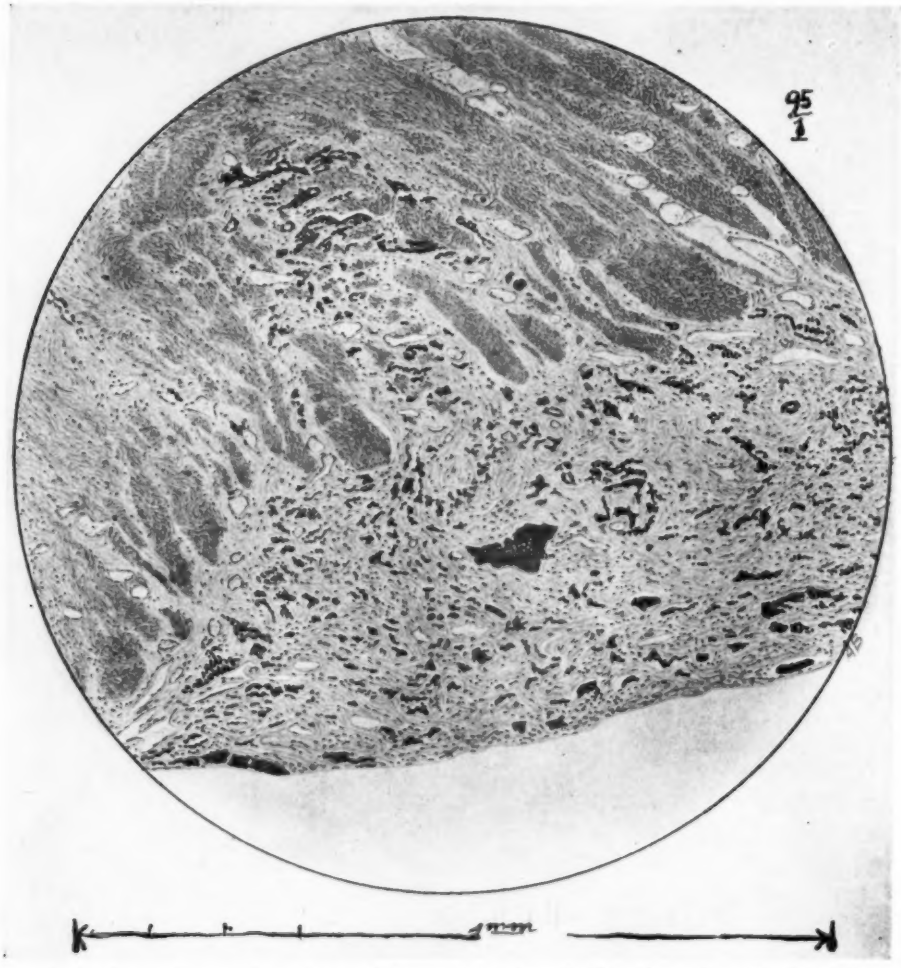

Fig. 4. Microscopical view of serosal surface of ileum. To show infiltration of carcinoid cells through muscular coat.

Carcinoid tumors which have metastasized have larger cells as a rule than the ordinary benign carcinoid tumors. Mitoses are never abundant. A number of thin-walled blood vessels are found within the tumor cell nests; this phenomenon of angiotropism is characteristic of carcinoid tumors and does not occur in true carcinomas. There is no true supporting stroma within the cell nests, but surrounding these groups of cells are fibrillar hyaline tissue and smooth muscle.

The muscularis mucosae is always present but is usually fragmented so that the cell masses are surrounded by many thin strands of smooth muscle. There is a variable degree of invasion of the muscular coats of the small intestine. This is another peculiarity of this neoplasm in that it is an infiltrating benign tumor. The serosa is rarely involved but occasionally is infiltrated as noted in the accompanying

case report (Fig. 4). The mucosa overlying the tumor is usually intact.

METASTASES

Stewart and Taylor were able to find only 18 cases of carcinoid tumors with metastasis recorded in the literature prior to 1926. Dukes and Lockhart-Mummery added another case. Ransom and Notthaft have also reported instances where carcinoid tumors of the ileum gave rise to metastases. There have been numerous instances reported where the carcinoid tumor cells have been found in the lumens of the veins. The direct extension of cords of tumor cells onto the serosa and into the mesenteric fat should not be construed as an indication of malignancy. When metastases do occur they are found most frequently in the regional lymph nodes, peritoneum, liver and pleura. Because of the rarity of metastasizing carcinoid tumors we feel justified in reporting our unusual case.

PROGNOSIS AND TREATMENT

The growth of carcinoid tumors is usually very slow. The majority of these tumors remain benign and harmless. Local excision is usually sufficient as they have no tendency to recur. Their growth is usually asymptomatic and the first indication of their presence may be intussusception. If metastasis has occurred to the regional lymph nodes, these should be removed at the time of the excision of the primary tumor.

CASE REPORT

J. S., a male negro, aged seventy-three years, was admitted to the Hillman Hospital, Birmingham, in a semicomatose state. No past or present history was obtained. The clinical diagnosis was cardiovascular-renal disease. The patient died in a few hours after admission.

At the necropsy, there were bilateral pleural adhesions and chronic pulmonary tuberculosis. There was generalized arteriosclerosis with very large ulcerated atheromatous plaques in the aortic arch. The atria were dilated and there was fatty infiltration of the heart. The kidneys were of the small red type which microscopically showed evidence of vascular nephropathy (arteriocapillary fibrosis).

The alimentary tract was normal except for the presence of 2 small sessile nodules in the lower ileum. The lowermost of these nodules was 9 in. distant from the ileocecal valve. These nodular tumors were situated 5 in. apart and there was no indication of a common origin. They projected into the lumen and caused a slight degree of stenosis. One of these tumors measured 1.5×2 cm., the other measured 1×1 cm. The mucosa was intact and surrounded the tumor. On cut section the tumors were of the same consistency throughout. They were grayish-white and waxy, firm yet elastic.

Microscopically, the intestinal villi were largely replaced with cords and nests of carcinoid tumor cells. In the superficial part of these tumors the cell nests were large and the cells had a tendency to form glands. But in the deeper portions of the tumors the nests and cords were smaller, there was no pseudoglandular formation and many of the cells were infiltrating the muscle layers in thin strands. The muscularis mucosae and connective tissue formed a stroma for the cell cords. Many small blood vessels were seen in the cell nests; they had no particular relation to any part of the cell nests, but were situated at irregular locations. The nuclei were large, pale and ovoid. The peritoneum was infiltrated with small strands of tumor cells.

Several enlarged lymph glands were found in the adjoining mesentery. The largest of these mesenteric lymph glands contained a large deposit of carcinoid tumor cells, which in some areas had entirely replaced the lymphoid structure. The diagnosis was confirmed by Dr. James Ewing.

SUMMARY

The various theories of the histogenesis of carcinoid tumors are reviewed. A case report is given of an unusual instance of multiple carcinoid tumors of the ileum with metastasis to a mesenteric lymph gland. [For References see Author's reprints.]

ROUTE OF METASTASIS IN CANCER OF THE BREAST*

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ENLARGEMENT of the axillary or subclavicular lymph glands is generally considered the first sign of metastasis in carcinoma of the breast. In over 60 per cent of cases the axillary nodes can be felt to be enlarged when the patient presents herself. In other cases, the glands are not enlarged but are found to be malignant when removed at operation.

The case I am reporting is of interest because it showed malignant involvement in a single group of glands, one that is frequently overlooked in surgical exploration. I am referring to the glands lying along the third digitation of the serratus magnus muscle.

Another interesting feature was the peculiar type of tumor, which resembled a multilocular proliferating cyst of the ovary. However, the epithelial cells showed unquestionable evidence of carcinomatous change.

With reference to the anatomical routes by which cancer spreads from the breast, the most frequent form of lymphatic dissemination is by way of the lymphatic vessels that empty into the lymph nodes overlying the digitation of the serratus magnus arising from the third rib. There are 2 anatomical reasons for the predilection of cancer metastasis from the breast for the serratus glands: (1) This region includes the great majority of mammary lymphatics; (2) it drains the lymph from the part of the breast which is most frequently involved in malignant disease, namely, the upper and outer quadrant.

The subclavicular glands and those in the axilla are only secondarily involved in cancer of the breast. Yet, as a general rule, they receive most attention in searching for metastasis and also in the general eradication of lymphatic tissue to provide against recurrence.

The anatomical facts and the findings in the case I am reporting support the belief that the glands lying along the third digitation of the serratus magnus are the first to be involved in malignancy of the breast. As a clinical deduction, especial care should be taken to remove these glands during the radical operation. Because of their deep situation below or along the lower border of the pectoralis minor, they are undoubtedly often overlooked and provide a source for recurrence.

CASE REPORT

Y. S., a married white woman, aged sixty-two years, was admitted to the Sydenham Hospital on November 19th, 1928, complaining of a lump in the left breast. It was first observed three months previously, since which time it had grown rapidly. The mass was located in the upper and outer section of the breast. There was no enlargement of the axillary glands or supraclavicular tension.

Because the rapid growth of the mass, which was slightly adherent to the skin, strongly suggested malignancy, operation was performed. A triple incision was made: (1) in the axillary region along the upper hair line, (2) along the lateral edge of the pectoralis major muscle and continued so as to surround elliptically the skin area of the breast with the tumor in its center, and (3) continued over the xyphoid process of the sternum.

The upper flap was reflected first and dissected upward as far as the lower portion of the clavicle and inward as far as the middle of the sternum. The lower flap was dissected downward as far as the anterior sheath of the left rectus muscle (epigastric triangle) and the fascia of the external oblique muscle; laterally, to the serratus magnus and latissimus dorsi. The sternal portion of the pectoralis

* Submitted for publication February 14, 1930.

major was severed at its insertion to the humerus and again at its origin from the ribs and sternum. The whole mass was turned outward and removed in one piece together with the fat and glands found in the axillary fossa. As careful examination showed no enlarged glands in the infraclavicular region, the pectoralis minor was not removed.

The operation was conducted with a minimum of hemorrhage. Bleeding of the larger vessels was arrested by hemostats; from the smaller ones, by hot compresses. Before the skin was closed, a soft rubber tube of the thickness of the little finger was introduced through a stab opening in the posterior flap immediately in front of the latissimus dorsi and placed in the axillary fossa along the course of the vessels in such a manner as not to compress them. The skin was sutured with silk. Duration of the operation fifty minutes.

During the operation careful search was made for enlarged axillary glands in the expectation of finding evidence of metastasis, but apparently the main group of glands was free from involvement. However, palpation in the region of the digitation of the serratus magnus arising from the third rib revealed a few enlarged lymph nodes, which were removed for examination.

Recovery was uneventful, and the patient was discharged from the hospital with the advice to undergo a course of postoperative radiation treatment.

On pathological examination a stony hard mass the size of a walnut, lying in the upper and outer quadrant about 2 in. from the nipple, was found in the excised breast. It was not attached to the underlying tissue. On section, the tumor was found to be cystic and exuded a dark brown fluid. The lining membrane of the cyst appeared reddish brown and hyperplastic. No other masses could be found in the breast.

Fig. 1.

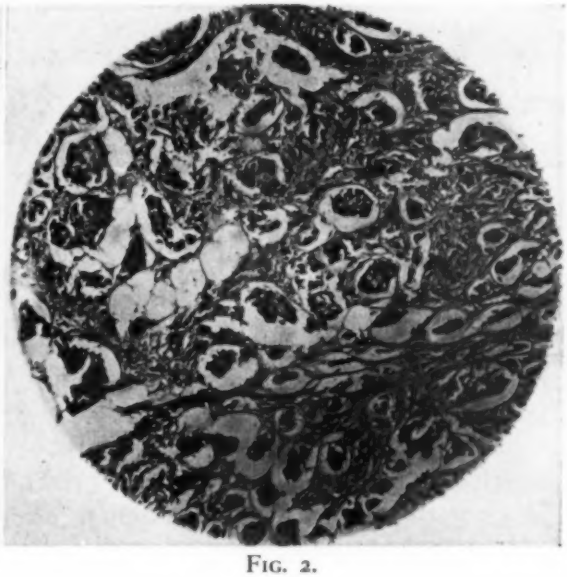

Fig. 2.

Microscopically, the growth showed numerous large cystic cavities, from the walls of which sprang numerous papillomatous proliferations composed of ill-defined anaplastic epithelial cells in all stages of mucoid degeneration. The entire histological picture closely resembled that of a multilocular proliferating papillary cyst of the ovary. In other sections, mucoid degeneration had advanced so far that individual cells could hardly be recognized.

Sections from the lymphatic glands removed from the region of the serratus magnus showed a large part of the lymph node to be replaced by cellular areas composed of large pale epithelial cells, which by contrast with the deeply stained surrounding lymphatic tissue appeared still paler. These cells were identical with those found in the original tumor. The axillary glands revealed no malignancy.

The pathological diagnosis was gelatinous cyst adenocarcinoma of the breast with metastasic colloid carcinoma of the lymph nodes in the region of the third digitation of the serratus magnus.

The pathological examination was conducted by Dr. A. A. Eisenberg, to whom I wish to express my best thanks.

PSEUDOPERITONITIS

DUE TO EXTERNAL TRAUMATISM*

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THE value of criticism lies not so much in uncovering mistakes as it does in finding means by which we may avoid future occurrences of similar mistakes. That many abdominal operations are performed and nothing found to account for the symptoms of which the patient had complained, is a fact well known to surgeons.

Articles that have been written in the past on the subjects of pseudoperitonitis, or peritonism, and pseudo-ileus deal, mainly, with medical conditions as the causative factors. Several cases have been observed by the writer in which the causative factor was a traumatism. In our present age of automobile and industrial injuries, there must be many instances in which accidents of a similar nature occur. It is with this in mind that an attempt is made in this article to clarify the diagnostic difficulty with a view to avoidance of unnecessary abdominal sections. One might say that no harm has been done by opening an abdomen under strict aseptic precautions, as practiced today, which may be true. Our contention is, however, that any refinement in diagnosis, which will help us recognize the uselessness of an abdominal operation in a given case, is a goal toward which every surgeon must strive. Not every section is entirely free from dangerous sequelae.

The first paper, dealing with these subjects, was written in 1865 by A. H. Henrot of Paris. Then follow papers on both subjects naming as causes: Intestinal ptosis, intestinal parasites, uremia, syphilis, hernia, ureteral calculi, pneumonia, instrumental delivery and adherent placenta, calcified mesenteric glands, heart disease, lead poisoning, diabetes, and hysteria. Nowhere in the literature have we been able to find mention of external traumatism as a cause of peritonism. We cite 5 cases and then attempt to formulate some plan of procedure in this type of case to enable us, if possible, to make a definite diagnosis.

CASE 1. A man, twenty-two years old, fell from a scaffold, 15 ft. high. He was immediately transferred to the Syracuse Memorial Hospital. He complained of severe pain in the left hypochondriac region. His skin was cold and clammy; pulse, 120; and respirations, 40. His only complaint was severe pain in the upper, left abdomen and lower back. The physical examination was negative, except for a marked rigidity of the upper, left rectus and tenderness, on pressure in that region and disclosed a swelling in the lower lumbar region of the spine. The man had fallen on his back, landing first in a half-sitting position and then rocked onto his neck, so the possibility of a fractured vertebra was kept in mind. Although at the time, the picture of an intra-abdominal lesion was not definite, consultation with the attending chief surgeon on the staff decided us in making an exploratory laparotomy. Careful examination of the stomach, transverse and descending colon, left kidney, spleen and small intestines disclosed no lesion. All organs appeared normal. The patient's temperature rose to 101°F., rectally, and in three days was normal. His pulse rate gradually receded from 120 to normal in the same number of days. He was discharged from the hospital twelve days after admission, during my absence from the city. Finding, on my return, that he had been discharged, I saw him at his home and had him return to the hospital for an *x*-ray of his spine because of my suspicion that we were dealing with a fracture. The posterior bulging in the lumbar region was definite, and the plates showed a compression fracture of the fourth lumbar vertebra. A plaster jacket was applied. He is well today.

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Case II. A child, four years old, was brought to the Syracuse Memorial Hospital following a coasting accident. He had a contusion and swelling of the soft parts on the left side of his neck, and a few skin abrasions of the face. He had no other marks on his body. His respirations were rapid and of a grunting character, such as one sees in irritation of the peritoneum in children. The abdomen, in its upper half, was held rigid. The possibility of a ruptured viscus decided us in opening the abdomen, but nothing was found.

Case III. An Italian laborer had a sudden pain in his upper abdomen, while lifting a door. After two days his physician sent him to the hospital with a diagnosis of acute cholecystitis; the surgeon, who saw him, concurred in this diagnosis. The blood count and temperature were normal. The upper abdomen, however, was markedly rigid and very tender. He had vomited once before admission to the hospital. A longitudinal incision was made. The gall bladder was normal, but a hemorrhage was noticed in the falciform ligament. When this was cut down upon, a clot, the size of an English walnut, was found; removal of this revealed active oozing. His convalescence was stormy because of hiccoughs for three days.

Case IV. A woman, aged thirty-eight, was brought to the Syracuse Memorial Hospital, having been crushed between an automobile and a tree. Upon admission she was found to have severe pain in the chest and abdomen, numbness and coldness of her legs, and was unable to raise her legs. Examination revealed market tenderness in the lower abdomen, considerable distention, and rigidity. Tenderness was very marked over the pelvis. *X*-ray examination of the pelvis showed a fracture through both acetabuli, also two fractures of the inferior ramus of the right pubis. There was practically no displacement of the former, but some of the latter two. Distention persisted for about *eight days, gradually subsiding in fourteen days*. Tenderness was a constant symptom. During the earlier part of her stay at the hospital, the patient was incontinent, but after twenty-seven days she gradually overcame this difficulty. At no time was her temperature over 100°F. No operation was performed.

Case V. A woman, twenty-nine years of age, came to my office. Her chief complaint was pain in the epigastrium and the left hypochondrium. This pain had been persistent for about one year and, at times, quite severe.

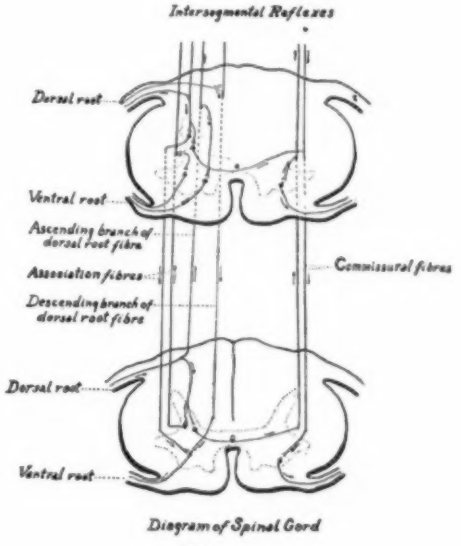

Fig. 1.

Examination disclosed definite tenderness over these areas, but it also extended along the course of the left, sixth, thoracic nerve to the postaxillary line. *X*-ray examination revealed no abnormality of the vertebrae or ribs. An inch of the sixth thoracic nerve was resected in the postaxillary line, and she has been free from symptoms since.

It is the purpose of the writer to explain how impulses may arise at one level; that is, an injury at the level of one vertebra may produce symptoms at a higher level through purely intraspinal connections. The diagram (Fig. 1) and explanatory notes will make this clear.

We are particularly interested in a description of an impulse, which will cause a reaction at a higher, sometimes much higher level than that at which the injury occurs, or in some instances, the opposite. One of the means through which this mechanism occurs, is the dorsolateral tract of Lissauer. In the diagram this is labeled "ascending branch of dorsal root fiber." It will be seen that the impulse enters the posterior root and then runs upward in the posterior funiculus of the cord, that is, in the white matter of the cord. It finally ends in one of the higher

segments of the cord and, in some cases, even in the gray matter of the brain. In this higher segment, the fiber forms a synapse in the gray matter of the cord with the motor pathway that goes to the area supplied by the emerging motor nerve in the ventral root.

Another means by which an ascending impulse may be conveyed is shown in the diagram. Following the arrows, entering the posterior root, it will be noted that there is a division of the fiber. The fiber running cephalad has been described. The one running inferiorly in the diagram is shown to divide again, and these divisions are shown to form synapses in the gray matter. It is the lateral of these two synapses with which we are now concerned. After the synapse, which takes place in the same segment, it will be noted that we again have a fiber running upward. This fiber is known as an association fiber and is so labeled. It will be seen that it makes a similar synapse in a higher segment, as the one already described in the tract of Lissauer; the final impulse is the same. In the diagram, a fiber is shown running parallel to the one just described, with the arrow pointing downward. The impulse conveyed by this fiber may run through several segments passing in the opposite direction, and through this latter mechanism, injury high up may manifest itself in an area below.

In the case of the fractured fourth lumbar vertebra, had we been dealing with a ruptured spleen or kidney, a blood count would have given evidence of hemorrhage by a lowering hemaglobin and red cell count. In cases of this sort, blood counts should be done not once, but several times in the course of an hour or two. Blood in the urine would have given evidence of a kidney lesion. A ruptured viscus would have given evidence of gas in the abdominal cavity, possibly absence of liver dullness. In addition, either of these conditions would be accompanied by marked shock. It is not necessary to go into further detail in the matter of physical findings in acute abdominal crises, which are the result of definite lesions to the viscera. They are well known and should be kept in mind by the surgeon. Case I had none of these findings, the only symptoms in common with direct peritoneal irritation were rigidity and pain. Had we known of the possibility of reflex arcs, such as described, causing this sort of disturbance, undoubtedly, we would have waited and watched our case for some time before doing a laparotomy, as we shall do in the future.

In Case II we had but two symptoms resembling peritoneal irritation; viz., a fairly rigid upper abdomen with apparent distention. Investigating this case from a physiological standpoint, we have been informed that it is quite possible that a blow of enough severity to irritate the phrenic nerve in the neck might cause a spasm of the diaphragm on the same side. The contusion in this case was over the area between the third and fourth cervical nerves, in other words, at the origin of the phrenic. It is not, in my opinion, beyond possibility that in this case our upper abdominal rigidity was caused by this injury. We are aware of the resemblance to peritonitis caused by a pneumonia with a diaphragmatic pleurisy. The child was sitting up in bed on the day following the operation and made, as Baldwin puts it, "a very uninteresting recovery." In another case of this type wait and watch will be the order.

Case III is included because of its unusual character, and because we felt that it fitted in with cases of pseudoperitonitis. It was the result of traumatism, and we were privileged to observe it. A hemorrhage into the falciform ligament of the liver, an entirely extraperitoneal structure, caused symptoms which, three men agreed, resembled cholecystitis. There was no rise in blood count, no history of indigestion; in fact, nothing definite except epigatric pain and one attack of vomiting.

Tenderness and pain from injuries in the abdominal wall may be differentiated

from deeper-lying causative factors; in that light palpation elicits pain, whereas it does not in deep-seated pathology. In this case there was aboslutely no evidence of peritonitis on inspection after the abdomen was opened. We cite this case merely to put it in the literature as another etiological factor in pseudoperitonitis. The stormy convalescence with hiccoughs shows that simple laparotomy is not always without danger.

Case IV is not an uncommon type and is mentioned to show how peritoneal irritation in pelvic fractures may cause distention, pain and rigidity resembling an "acute abdomen." Nothing was done in this case in an operative way. This woman made a fine recovery in all respects.

Case V is a very interesting type. Subcostal neuralgia has been spoken of as a cause of symptoms resembling those due to pathology of internal organs. Cases have been cited in which several abdominal operations were performed, relief finally being obtained by section of the nerve. In our case, before the nerve was sectioned a 2 per cent novocaine solution was used as a block anesthetic. This was injected around the nerve, posterior to the angle of the sixth rib. The patient immediately stated that the pain had stopped. Palpation revealed no area of tenderness in the epigastric or left hypochondriac regions, or along the course of the nerve. Here then we had proof that section would relieve the pain. This was done with, until now, gratifying results.

This last result has given us the impression that, possibly, in cases of the type found in Cases I and II, a block anesthesia along the peripheral nerve or nerves supplying the segmental area affected, may aid in the differential diagnosis between traumatic pseudoperitonitis and actual visceral damage. In case the pain is relieved following the injection, and no pain or tenderness develops in another area, such as we see, for instance, in a case of perforated, gastric ulcer, where the pain may gradually go downward from the epigastrium to the appendiceal region due to peritoneal irritation of the gastric contents running down the right gutter, we may presume that a viscus is not injured, and that we may safely wait. Naturally, blood pressure, blood count, temperature, etc. would be carefully noted meanwhile, and noted frequently.

SUMMARY

1. The type of case with which this paper deals is one which demands the minutest and most painstaking personal care of the surgeon himself.

2. The decision whether to operate or not to operate must be made in a matter of a few hours.

3. Keeping in mind the possibility of distant traumatism as a factor in the causation of symptoms resembling those due to actual peritoneal inflammation, it but remains for the surgeon to stay with his patient and to carry out meticulously all the essential clinical investigations, which will show actual peritoneal injury.

4. Use of block anesthesia during this time is suggested as an aid in the diagnosis.

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CALCULUS OF THE UPPER URINARY TRACT*

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We wish to present certain data concerning 70 patients with calculus of the upper urinary tract, operated upon at Bellevue Hospital by the staff of the Urological Service; also to state briefly our present attitude toward certain problems encountered in this work.

RENAL CALCULUS

There were 164 patients discharged from the hospital the past six years with the diagnosis of nephrolithiasis. Forty-eight of these were operated upon and form the basis of this review. Three were readmitted for operation on the opposite side.

Our wards always contain a relatively large proportion of individuals of foreign birth, and it is therefore not surprising that 11 nationalities were represented in this group. About one-third were born in the United States and an equal number in Italy. As to the age incidence, our patients were about equally divided below and above forty. Our youngest case was twenty, and the oldest seventy-three. Hospital rules prohibit the admission of children under fourteen years of age in the Urological Service.

Complaint: The most frequent complaint was loin pain, noted in 80 per cent, while abdominal pain was recorded in 15 per cent of the cases, urinary frequency in 12 per cent and hematuria in 10 per cent. Occasional complaints were anuria, foul-smelling urine, dysuria, loss of weight, weakness, nausea and vomiting, headache, testicular pain and multiple arthritis.

Past Urinary History: Thirty per cent only had had previous ureteral colic; 7 per cent previous attacks of hematuria; 10 per cent had passed a stone; nearly 15 per cent had had previous operation for calculus; and 12 per cent (all with right renal stone) had submitted to appendectomy, quite possibly because of ureteral pain.

Examination: Tenderness in the kidney area posteriorly was found in nearly 60 per cent of the cases. Abdominal tenderness occurred in about 30 per cent. The renal function on the diseased side in unilateral stone cases as compared with the other kidney, was determined by the urea content of the urines, and the output of indigo carmine and phenolsulphonephthalein. It was found that the function was decreased in 70 per cent of the kidneys harboring stones. Curiously in 3 instances the function as indicated by these tests was really increased. Infection of the urine obtained from the calculus side was present in nearly 85 per cent. The pyelograms showed some dilatation of the pelvis or calyces in 95 per cent but rarely if ever the irregular outline of kidney destruction.

Of the bilateral stone cases, we call particular attention to 3 patients, whose output of phthalein in two hours before operation was surprisingly large, 65 per cent, 50 per cent and 50 per cent respectively. In these patients, the complaints demanding operation were renal pain in all, chills and fever in 1, and hematuria in 1.

ILLUSTRATIVE CASE REPORTS

For contrast, the following 2 interesting cases of bilateral calculus disease are cited.

Case 1. E. B., male, aged forty-eight, entered the hospital complaining of pain in both flanks. He had had a pyelotomy for stone fifteen years earlier. He had been under the observation of one of us for five years, and had sought advice because of cloudy urine. Ureteral catheterization had repeatedly demonstrated infection of both kidneys, and the phthalein report was excellent, although roentgenograms showed stones on both sides

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(See Fig. 3). But there were no symptoms and the general health was good over this period. Finally chills and fever developed,

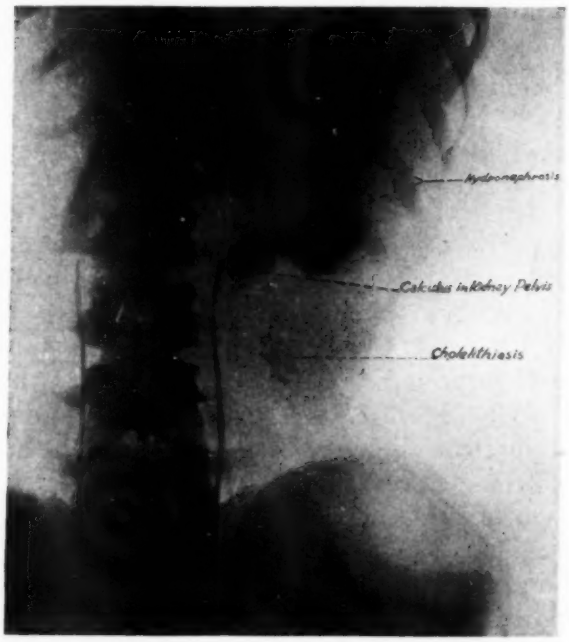

Fig. 1. Calculus in renal pelvis producing hydronephrosis. Eleven stones in gall bladder.

with severe pain in both kidney regions. On admission to the hospital, the patient's phthalein output in two hours was 65 per cent. The right kidney gave but a trace of urea, no indigo carmine, but some pus and micrococci; the left kidney gave 0.5 per cent urea, blue color in eight minutes, pus and micrococci. Left pyelotomy and nephrotomy were done for multiple calculi (Fig. 4). Phthalein output on discharge was 25 per cent. On readmission, four months later, it was 30 per cent. The right kidney gave no urea, no dye, but much pus and cocci; the left kidney gave 0.3 per cent urea, pale blue color, clumps of pus and some cocci. Right nephrectomy was performed for advanced calculus pyonephrosis. Two weeks after operation, the phthalein was 40 per cent in two hours. Patient continues in good health to date, eight months after operation.

Case II. C. A., male, aged thirty-two, entered hospital complaining of pain in the left loin, foul urine, headache and nausea. Non-protein nitrogen in the blood plasma was 145 mg. per 100 c.c., and creatinine was 3.6. Cystoscopies showed thick ribbons of pus from both ureters; there was no phthalein output in eighteen minutes, and no indigo carmine in twenty-five minutes from either side. Roentgenograms proved calculi in both kidneys. The usual medical therapy was persisted in but availed nothing; surgery in our opinion was contraindicated. The patient became gradually worse, with vomiting and other evidences of toxemia and died on the seventeenth day. N.P.N. three days before

Fig. 2. Multiple calculi in one kidney. Kidneys show equal function and no inflection. Yet for thirty-seven years patient has had occasional hematuria, especially after exercise, without pain. Operation not advised, as condition good and patient comfortable.

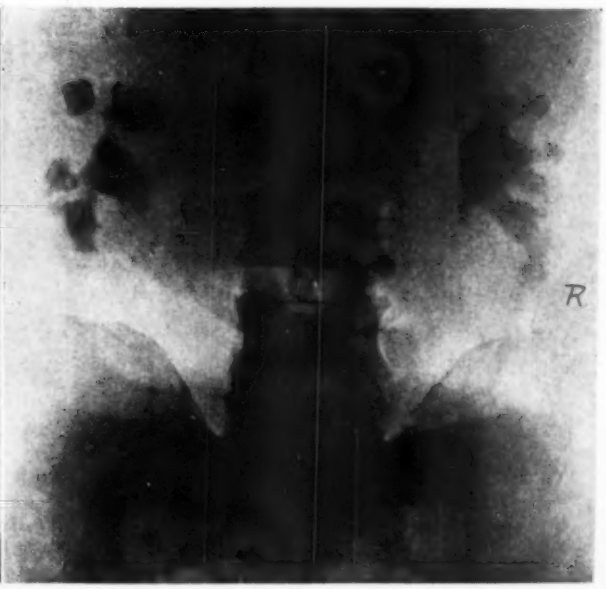

Fig. 3. Bilateral calculi. Multiple stones removed from left kidney. Later right nephrectomy for calculous pyonephrosis.

death was 132, creatinine 4.0; one day before death, N.P.N. was 282 and creatinine was 5.7.

Anesthesia: In the earlier cases of the group here reported, general anesthesia was used. Having used spinal anesthesia with increasing satisfaction in perineal and suprapubic work the past ten years, we were led to try it in kidney and ureteral operations, as the technic had improved and the safeguards multiplied. In 1928 spinal anesthesia was used in 11 kidney operations. Three of these, lasting over one hour, required some general anesthesia at the close. Contrary to the opinion expressed by some anesthetists, we find that patients under spinal anesthesia take the added general anesthesia with particular ease. Spinal anesthesia gives marked muscular relaxation during the operation, leads to less postoperative distention, less gastrointestinal upset and fewer respiratory complications. For routine work, we believe it to be the method of choice. However, it must be employed only with a thorough knowledge of the technic and dangers. The great risks are anesthesia of the medullary centers and rapid fall of blood pressure. To combat the former, the spinal puncture is done with the patient lying on one side, and the head is kept lower than the hips during and after operation. To combat the latter, we depend almost solely upon ephedrine. Uniformly a subcutaneous injection of 50 mg. is given at the time of the spinal tap. An interne is detailed to watch the blood pressure, and further injections of ephedrine, subcutaneous or even intravenous, are given as indicated. However, spinal anesthesia is not for universal application. There are some emotional and apprehensive patients who are constitutionally unfitted for it.

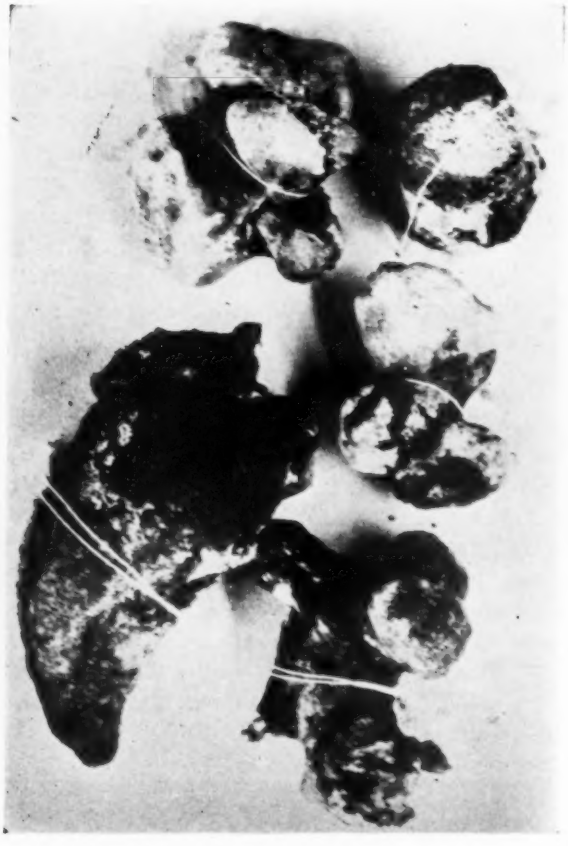

FIG. 4. Stones removed from left kidney of case shown in Fig. 3.

Operation: An oblique loin incision parallel to the costal margin and keeping above the twelfth dorsal nerve, is almost always employed by us. In some cases, the stone is easily felt in the pelvis or upper ureter, and readily removed without shelling the kidney from its fatty capsule. We believe this should be done when feasible. Otherwise the kidney is freely mobilized and brought out of the wound if the length of the pedicle permits. It seems universally agreed that pyelotomy is preferable to nephrotomy. When incision of the renal cortex is necessary we prefer to extend the pyelotomy wound into the kidney parenchyma upward or downward as the position of the stone indicates, carefully avoiding visible vessels. Any bleeding vessel may be carefully clamped and tied. Often such hemorrhage is quickly and is preferably controlled by pressure. These kidney incisions are closed by a few superficial mattress sutures placed in the capsule. We have avoided deep through and through sutures as tending to cause pressure necrosis, at times additional hemorrhage, and postoperative infection. This superficial suture

has completely controlled the bleeding. When isolated calculi are present in calices, it may be necessary to cut the convex surface of the kidney parenchyma. We prefer multiple small incisions to one large one. After doing nephrotomy, we always incise the pelvis and leave it open for drainage. There were 10 cases requiring nephrotomy in this group. None had subsequent hemorrhage nor any secondary operation.

Fig. 5. Tuberculous calcification in lower calyx of kidney resembling renal calculus.

Fig. 6. Calcified lymph node outside middle calyx.

It has been a question with us for some time whether suture of ureterotomy or pyelotomy wounds hastened the healing. Some of these cases without suture have never drained urine, and we are using sutures in decreasing numbers. At present only the larger incisions are sutured and they only partly so. If there is no obstruction, leakage from a ureteral incision will be little; the normal peristalsis carries urine toward the bladder. If there is some permanent or even temporary obstruction, it is much better for the patient that an opening in the ureter be left.

Wounds: No clean wounds in this series, that is, wounds leading to uninfected kidneys, became infected. All wounds but 3 of 15 pyelotomy cases with definite notes, healed flush with the skin within twenty days; 2 in twenty-five days. One case leaked urine forty-two days; this healed only after several indwelling ureteral catheters had been used.

The nephrectomy wounds healed on an average of one week earlier than the pyelotomy wounds.

Of the whole series of kidney cases, 4 wounds had to be opened because of infection.

Stay in Hospital: About 40 per cent of the patients left the hospital fifteen to nineteen days after operation; 40 per cent in twenty to twenty-eight days; and 20 per cent remained in hospital over four weeks.

Deaths: Of the 51 patients having renal calculus who were operated upon, 8 died. Believing that our failures are of particular interest, we record a few facts concerning these 8 deaths. All of them occurred within two weeks after operation; 2, within twenty-four hours. Perhaps it is only fair to state again that this series of operations for renal calculus with such a high mortality rate concerns only patients of a municipal hospital. A large percentage of these patients are admitted in very bad condition, and circumstances frequently compel us to take grave risks (Note Cases III, VI, VIII–X following).

Case III. Male, aged sixty-three, had a

large calculus removed through a pyelotomy incision. He was in a weakened condition with a poorly functioning heart, but operation had to performed because of continued pain. Condition satisfactory immediately after operation, but patient became gradually weaker and died of myocarditis two weeks after operation.

Case IV. Male, aged thirty-three, was operated upon for stones in the renal pelvis. The pedicle was accidently torn during the dissection, severe hemorrhage ensued necessitating nephrectomy. Patient died in twelve hours.

Case V. Male, aged forty-seven, had a nephrotomy for calculus. Previous cystoscopy showed no infection of either kidney. Condition satisfactory until the thirteenth day when the patient suddenly became cyanotic and died. The attending physician diagnosed cornary embolism. No autopsy.

Case VI. Male, aged fifty-nine, had right nephrectomy for calculus pyonephrosis. General condition poor before operation. N.P.N. was 60 and creatinine 2.5 four days before operation. Phthalein tests 20 per cent in two hours one day before operation. Because of pain in the kidney region and infection, operation was imperative. Condition improved little after nephrectomy. On the tenth postoperative day, there was a chill with temperature 101°F., and severe sharp pain in the right lower quadrant. Patient died the next day; no autopsy. Probable cause of death was thrombosis.

Case VII. Male, aged thirty-four, was admitted with bilateral renal calculi, a large dentritic stone on the right side, and a large calculus in the left pelvis. On cystoscopy, the right ureter gave thick pus, no urea, no dye; the left ureter, no pus, good urea, and phthalein in seven minutes. Under spinal anesthesia, the very adherent right kidney was removed. Temperature after operation registered from 101 to 104.6°F., the pulse was rapid and abdomen markedly distended. Distention could be relieved only temporarily, and patient died on fifth postoperative day. No autopsy.

Case VIII. Male, aged fifty-four, entered hospital with pain in right loin and hematuria. Roentgenograms showed stones in both kidneys. On cystoscopy, right ureter discharged thick pus, and left ureter clear urine; no indigo carmine came from either kidney in eleven minutes. N.P.N. ten days before operation was 56; creatinine 3.0. N.P.N. five days before operation was 100, creatinine 3.0. Perinephritis abscess became apparent and was drained; no further operation done. On second postoperative day, N.P.N. was 300, creatinine 4.3. Condition steadily grew worse, and patient died on seventh day after operation. No autopsy.

Case IX. Male, aged forty-three, was admitted with severe pain in the right loin, vomiting and headache. He had been operated upon for stones in the right kidney two years earlier, and had had a litholapaxy a year before that. N.P.N. a week after admission was 200, creatinine 2.6; twelve days later, N.P.N. was 30, and creatinine 2.2. No cystoscopy, the right kidney gave a faint trace of indigo carmine and 0.65 per cent urea; left kidney, medium color and 1.1 per cent urea. There was some infection on both sides. Because of the poor condition, no operation was done until six weeks after admission. However the pain continued and operation seemed imperative; the right kidney was removed under general anesthesia. Operation was not unduly prolonged, yet the patient went into shock on the table and died the next morning. No autopsy.

Case X. Male, aged fifty-three, was admitted with a history of anuria for twenty hours. Was relieved by indwelling catheters for three days when he insisted on going home. He was able to urinate for four days, then developed trouble again and returned after three days of anuria. Double indwelling ureteral catheters were introduced at once. The next day N.P.N. was 84 and creatinine 4.5. Five days later, (the catheters having been withdrawn), the N.P.N. was 230; the catheters were reintroduced. Several phthalein tests done at various times gave zero output in two hours. Roentgenograms showed small shadows at the uretero-pelvic junctions on both sides. From time to time the ureteral catheters were withdrawn; patient would become anuric and uremic, and catheters would be reinserted. Finally on the twenty-fifth day after admission, N.P.N. was 56 and creatinine 3.2. Operation offered the only hope of permanent relief, as otherwise better condition was dispaired of. Right pyelotomy was finally

performed and stone removed. There was considerable infiltration of the perinephritic fat noted. Patient died three days later. Autopsy showed a small right kidney with perinephritis; calcareous incrustations and thickened wall of the corresponding ureter, 2 cm. below the pelvis. The left kidney was large (25 cm. long) and had purulent material in the pelvis; at the uretero-pelvic junction there was a calcific deposit obstructing the ureter.

URETERAL CALCULUS

There were 153 patients discharged from the hospital during the past six years, diagnosed "ureteral calculus." Of these, 22 were operated upon by our staff.

As with our patients having renal calculus, over half of this group of 22 ureteral cases were born in this country or Italy. Considering their ages, 9 were in the fourth decade of life, 6 in the third, and 6 in the fifth decade, while but one was over fifty.

Complaint: Pain was the outstanding symptom; in 12, it was referred to the back; in the other 8, to various parts of the abdomen (1 to the penis). Hematuria was complained of but twice, anuria once and urinary frequency once.

Past History: Pain had occurred on the same side which was the seat of the present trouble, in 5 instances, from one-half to eight years earlier; hematuria, once, two years before; stone passed from the same side, once, two and one half years before; ureterotomy, once, eight years before; appendectomy on side of present trouble, once, eight years earlier.

Present Illness: Eighteen cases had pain in the kidney region; 3 had unilateral abdominal pain. Of these 21 patients, 8 had radiation of the pain to the groin or testicle.

Examination: There was costovertebral tenderness in 15 patients, and abdominal tenderness in but 3, while 6 patients presented no tender area.

As the general phthalein test was usually good before operation, there could be no appreciable improvement thereafter; no case showed a decrease of output after operation. The only interesting observation on blood chemistry concerned a patient with bilateral ureteral calculi, causing almost complete anuria. His N.P.N. jumped from 55, five days before operation, to 137 on the day of operation, creatinine going from 1.5 to 4.5. Four days after double pyelotomy both constituents were normal.

Catheters were made to pass the ureteral stones in all cases but 4. The renal function on the involved side was decreased in 14 of 15 cases where ureteral specimens were obtained; one showed no decrease. The function on the involved side showed definite improvement in all of 5 cases tested after operation. There was demonstrated dilatation of the urinary tract above the stone in all of the 4 patients in whom clear pyelograms were obtained.

Infection was present on the involved side in 11 of the 15 cases. After operation, the infection was absent in 2 cases cystoscoped, decreased in 1, and unchanged in 1. The uninvolved kidney was found infected twice and uninfected in 16 cases.

Operation: Incisions naturally varied with the location of the stones. They were high, practically as for a kidney operation, and low (over pelvic ureter) in about equal numbers. The low incisions were usually oblique, following in general that described by Gibson. In 4 instances the vertical mid-rectus incision was employed and found very satisfactory for stones near the bladder. However, we believe the oblique incision affords an easier approach in the larger number of cases. The stones were found and removed in all but 2 instances. In one of these the calculus slipped back to the kidney, was not found in the renal pelvis at this time, but was removed from the ureter at a later date. The other failure concerned one side of a patient in almost complete anuria from double ureteral calculi. We had one experience of failure to secure a stone low in the ureter, but opened the bladder at once

and obtained it from the intramural portion.

Ureteral stones sometimes change position markedly without causing pain. Hence we uniformly require that a confirmatory roentgenogram be made just prior to operation. At operation, ureteral calculi do not easily move down the ureter, but may readily slip up toward the kidney. It is a useful procedure to dissect above the stone as early as possible and place a tape under the ureter above the stone.

As stated earlier, we place sutures in the ureter only in instances of long incisions. Adequate drainage is obviously necessary.

Our preference for spinal anesthesia has already been stated, and it has been employed with great satisfaction in these cases for nearly two years.

Wounds: Two cases had no leakage of urine from the wounds. The others leaked for varying periods up to twenty-six days. If leakage continues after ten to fourteen days, a catheter is passed up the ureter and allowed to remain for a day or two.

Stay in Hospital: In this group of 22 operations for ureteral calculus, there were no deaths. Three (14 per cent) went home in eighteen days after operation; 11 (50 per cent) in twenty to twenty-nine days; 8 (36 per cent) in thirty to forty days. The last group represents chiefly badly infected wounds, the infection having been present in the corresponding kidney before operation. One patient had probable femoral thrombosis, but was discharged on the thirty-sixth day. One case with postoperative epididymitis was discharged on the thirty-second day. Apparently, patients stay in the hospital longer after ureteral operations than after kidney operations for calculus.

BILATERAL CALCULUS

The problem of bilateral calculus disease of the upper urinary tract has often been discussed. The usual advice is to operate first on the side having the better kidney. But some will decide this question on the basis of the comparative function tests of the kidneys as found before operation. This may be very misleading. The better kidney may be functioning badly for the time being, because of ureteral obstruction. It is necessary to consider the position of the calculi and to note the possibilities of obstruction, as this factor is the most potent one for immediate harm. Hence our feeling is that each case presents a problem unto itself. Having studied the patient, the cystoscopic and functional reports, and the roentgenograms, we advise operation first on that stone, the removal of which will enhance the total renal function the most.

CALCULUS ANURIA

There are differences of opinion concerning methods of treating patients with complete blockage of one or both ureters. Prolonged trial of palliative measures is to be condemned. The patient should be placed in a hospital where operation can be performed at a moment's notice. Cystoscopic methods will often relieve the obstruction, at least temporarily, will thus allow time for further study, and will put the patient in better condition should operation become necessary. Furthermore, in some instances, a flow of urine will become established, after the ureteral catheters have been removed, and the patient may ultimately pass the stone, if small. Continuous observation is urgently required, with frequent estimations of renal function. If this decreases, or the anuria recurs after the removal of the ureteral catheters, immediate operation is imperative. If the condition remains good, but the stone does not move, operation will probably be indicated, but the emergency has passed.

INDICATIONS FOR OPERATION

We wish, in closing, to consider briefly which stones in the kidney and ureter should be operated upon, and which should be left alone. As in all realms of surgery, one must fairly consider whether

the probable benefit to follow operation will compensate for the possible harm and risk.

Operation is usually indicated on kidney stones associated with reduced renal function; coincident infection increases this demand. The chances of eliminating infection accompanying stone without removal of that stone is slight. The stone should be removed through a pyelotomy incision if it seems feasible to do so. If the stone is small and so placed in the calyx that nephrotomy will be required, it may be questioned whether such an operation will not do more harm than good. Either persistent pain or hematuria is an indication for operation. Silent stones without infection, hematuria, pain, or diminished renal function, we think should be left alone.

As for ureteral calculi, we agree with the general view that all these stones should be ultimately removed, and that stones casting shadows 1 cm. or more in diameter will probably require operation. We have had patients pass such large stones, but this is not the rule. Smaller stones can usually be made to come away by cystoscopic manipulations. There is danger in prolonging these efforts; irreparable damage may ensue. Diminished function of the kidney, especially decreasing function and most particularly anuria, demand an early operation.

SUMMARY

1. We present data concerning 70 patients operated upon at Bellevue Hospital for calculus disease of the upper urinary tract; these represent 51 kidney operations and 22 ureteral operations.

2. The vast majority of our patients come to the hospital for the relief of renal pain.

3. Kidney and ureteral stones usually decrease the renal function, and increase any existing infection.

4. Spinal anesthesia has been used with great satisfaction in the more recent operations.

5. Pyelotomy is the operation of choice for kidney stones; a pyelonephrotomy or a nephrectomy may be necessary.

6. Only the larger pyelotomy and ureterotomy incisions are sutured after the removal of the stone.

7. Following the 51 operations for stones in the kidney, there were 8 deaths, most of which occurred in desperately ill patients with trouble of long standing and with symptoms demanding operative relief. There were no deaths following the 22 ureteral operations.

8. A curved loin incision is used for kidney and upper ureter stones; the oblique Gibson incision for lower ureter stones.

9. In cases of bilateral calculi, as a general rule operate first upon the stone, the removal of which will most markedly enhance the total renal function.

10. Indications for operation are, diminishing function, increasing infection, anuria, ureteral stone over 1 cm. in diameter, persistent pain, and recurring hematuria.

11. All ureteral stones should be ultimately removed by cystoscopic manipulations preferably, or by operation.

GONORRHEAL INFECTIONS OF THE BLADDER & KIDNEYS*

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As far as the bladder is concerned, it may very definitely be stated that only one part of it is ever involved in a gonorrheal infection. The region affected is a triangular area in the bladder neck, known as the trigone. It extends along the bladder floor from the orifices of the ureters to the internal urethral opening.

Its structure is practically identical with that of the urethra and the ureters. In all its other portions, the bladder structure is very different and apparently non-suspectible to gonococcal attack. We can thus eliminate bladder disease per se in gonorrhea. When a deep infection of the urethra is present, as it invariably is in all cases, the trigone also carries the burden of the disease. In these cases, however, there is much greater danger of the infection passing up into the kidney, than there is of its going over the trigone border into the bladder proper.

It has always been our impression that considerably more gonorrheal disease of the kidney exists than we realize, but a careful survey of living and autopsy material has failed to confirm this belief. Throughout a period of almost thirty years we have encountered only one case in our own practice, although we have seen 4 in the service of other physicians. The entire literature shows but 27 cases reported. Are these cases the result of a systemic or an ascending infection? We are inclined to believe that they are part of a so-called gonorrheal septicemia or blood poisoning.

Simmons a few years ago surveyed this situation in a very thorough way. The possibility of infection reaching the kidney immediately suggest two likely ways: the blood or via the urinary system. During the course of most acute infectious diseases, bacteria have been proved to enter the blood stream.

This has been shown repeatedly in the case of the gonococcus. The frequent gonorrheal inflammation of the heart and so-called gonorrheal rheumatism demonstrate not only that the gonococcus enters the blood stream but also suggest in a very modest way the frequency with which it does so.

It has been demonstrated also that bacteria may readily pass from the infected blood stream through the normal kidney without producing disease. If these bacteria are few in number or of the less violent type the healthy kidney does not become infected. If, however, they are abundant and of a violent strain, or if there exists a defect in the kidney itself, or one resulting in the stagnation of urine in the kidney pelvis, an infection of that organ will occur. As these conditions are rarely fulfilled, in an attack of gonorrhea, the infrequency of gonorrheal kidney complications is probably thus explained.

Simmons further states that the urinary route, upon superficial consideration, suggests the more likely source of kidney infection. However, when the defensive mechanism of the urinary system is considered, this route must be of secondary importance particularly in gonorrheal infections. The usual port of entry for the gonococcus is the anterior urethra. By direct extention, in a certain number of cases, the infection reaches the bladder neck, as we have shown. The ureter, however, seems doubly protected against further invasion. These are very important points in medico-legal cases. The ureter is first protected by the sphincter action of the bladder wall or the ureter. As a

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matter of fact there is really a fold of tissue forming a valve at the entrance of the ureter into the bladder. This fold holds so strongly that unless it is diseased we cannot force fluid up the ureter by over-distending the bladder. A second preventive barrier is the continuous flow of urine from the ureter into the bladder.

Any inflammation starting in the ureter at the bladder would also of itself tend to prevent an extension upward of the disease. Several other routes have been proposed for kidney infection. One is by a special utero-renal circulation demonstrated by Sampson, but this in reality does not actually touch the kidneys. The remaining source of infection is by the lymphatic channels. At this time there is a connection between the lymphatics of the bladder and the kidney, but it is interrupted so often that it does not seem a likely channel. I think the only case of record that could definitely be linked with lymphatic extension was an instance of multiple gonorrheal abscesses reported by Stanton in 1913.

To obtain definite proof that we have a gonorrheal infection of the kidney, the organisms must be demonstrated in the pus or tissues. Simmons says that of the 25 cases in the literature up to 1913 in only 15 was the gonococcus actually found. This, we feel, reduces the number of cases considerably, as many other organisms will give rise to symptoms of kidney infection. Sixteen of the cases reported in this group were males, one occurring ten days after the original infection in the urethra. The case reported by Stanton developed nine years after the original gonorrhea. This, we know, must be annoying for a gonorrheal patient to ponder over.

AUTHOR'S CASE

There is so little data available for study in the cases reported to date, that we believe you will be interested in first-hand information of our own case.

The patient was a young white woman, twenty-two years of age, a native of the United States. Her previous family and personal history are of relatively little interest, with one exception: she denied any possible venereal infection.

Present Illness: We were called to see this young lady who was a patient in the hospital in September 1928. Two weeks previous to our visit she had had an abortion performed, apparently at about the fourth month of pregnancy. On the following day she developed chills and fever and gradually a mass appeared over the left kidney. When we saw her, the mass was so large that it could be seen as well as felt. The patient was thoroughly septic and her condition was wretched. We suggested a vaginal examination, which for some reason she did not wish; but, by insisting, we finally obtained her consent to a complete observation. Almost before we had entered the vagina, we found a huge pelvic abscess.

It was at once evident that the primary source of infection was not in the kidney but in the lower genital tract. The pelvic abscess was first opened through the vagina, and a large amount of greenish pus was evacuated. Cultures from this material showed gonococci in abundance. Next, in the same sitting, we incised the swelling over the kidney. Upon passing through the muscle planes of the back, found ourselves in a large abscess cavity with the kidney in its center. In fact, the organ was so close to the surface that we saw almost at once that our abscess was connected directly with it. An opening into the kidney was found and cultures were taken from both it and the surrounding areas. All of the cultures and slides showed gonococci.

Further study of this case revealed that this young unmarried woman was probably infected with gonorrhea at the time of her impregnation.

You will say "what are the signs of a gonococcal kidney disease?" There are none whatever that are characteristic. One sees merely the symptoms of a kidney infection or abscess in or around that organ.

POSTERIOR COLPOTOMY FOR PELVIC INFECTION*

(FRAENKEL TECHNIC)

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OUR chief interest in pelvic infection is not in its frequency or in its chronic course, but rather in its tendency to recurrence and its close association with sterility and ectopic pregnancy, thus making it one of the most important diseases of the female genital tract. There are three general types of treatment:

1. Radical therapy consisting in laparatomy with removal of a major portion of the female genital tract.

2. The expectant therapy: rest in bed, care of the bowels, diathermy and foreign protein injections.

3. A course between the two, one not quite so radical as the first method and yet making full use of all conservative measures at our disposal.

There are two main objections to the radical form of treatment:

1. Due to the tremendous amount of infiltration and an absence of lines of demarcation of the disease it is frequently necessary to sacrifice a major portion of the female genital tract and as these conditions occur mainly in young adult life, a loss of both ovaries, tubes and part of the uterus makes the patient an invalid for life.

2. The mortality of this process is high and its morbidity very severe.

There are two main objections to the conservative treatment:

1. If it is carried too far, complications such as a rupture of the pus sac into the bowel, or rupture into the general peritoneal cavity followed by general peritonitis with its severe morbidity and occasionally death may occur.

2. Pure conservative therapy requires a long period of hospitalization which, besides the tremendous expense incurred, also means a distinct monetary loss to the patient due to her loss of earning capacity.

The mortality of the radical procedure being so high and convalescence from the purely conservative treatment so protracted, a path was sought between the two: one which would drain the infiltrated area and shorten the convalescence of the patient, thereby returning her to her economic state in the shortest possible space of time.

For this purpose, posterior colpotomy is an almost ideal method. This procedure has been in vogue for a great many years. Morely[1] states that "according to Bourdon,[2] Callisen[3] (1798) was the first to venture opening a pelvic abscess by the vaginal route. This procedure was copied by Macarn in 1788, who reported 3 cases." Since then, Dumarquay[4] (1857), Chassaignac[5] (1859) and Nonat[6] (1874) in France, Spencer Wells[8] in England, D. W. Brickell[10] (1877), Th. A. Emet[9] (1880) and Pryor[13] (1899) in America, all performed posterior colpotomy for the drainage of collections of pus in the cul-de-sac. Pryor even went a step further, opening the cul-de-sac in all pelvic infections where he suspected the possibility of pus forming. D. Warren Brickell[10] stated in 1877:

> My conviction is that if early operative procedure is once admitted to be proper (and for this I strenuously contend), the operation per vaginam will take complete precedence. I am satisfied that it must be rarely exceptional to find the purulent collection early "pointing" or inviting the knife or trocar towards the external surface. Long before this stage and when the abscess is comparatively small, puncture per vaginam will promptly relieve. There is a positive demand to remove serum as to remove pus; nevertheless it would be almost a hopeless task to convince any con-

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siderable number of surgeons to resort to such practice without practical illustrations to support the theory.

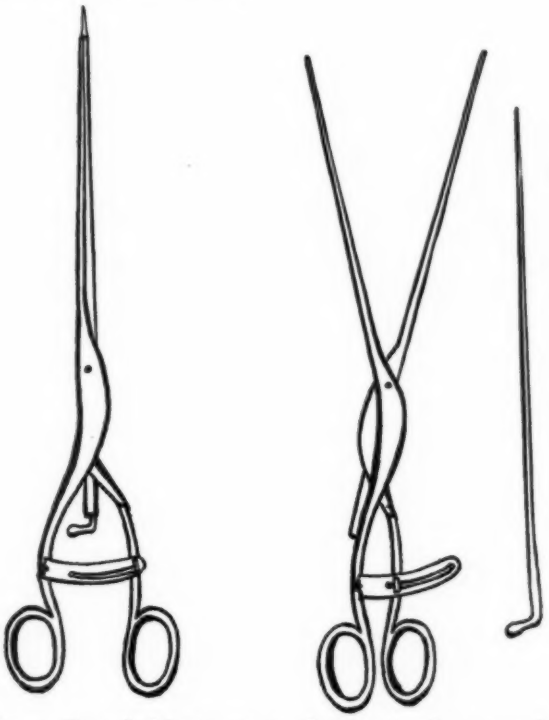

Fig. 1. Fraenkel instrument, showing trochar cannula and spreading blades.

Chassaignac[5] in 1859 gave the following indications for posterior vaginal drainage: vagina, and that vaginal fluctuation is definitely present. If the incision is too small to establish a free drainage, the opening may be enlarged with the aid of a bistouri boutonne.

The indications are practically the same today. Crossen[11] (1923) states: "If a collection of pus can be felt low in the pelvis, open and drain by vaginal incision." Eden and Lockyear[12] (1920) state: "When a cystic swelling forms in the Pouch of Douglas, it should be at once evacuated by posterior colpotomy."

In other words, 2 factors are necessary: (1) the development of a large fluctuating tumor, (2) the fluctuating tumor must present itself on the posterior vaginal wall. These 2 factors have prevented this procedure from being carried out very often because the 2 indications were not always met. One frequently would find a large nonfluctuating mass without the production of any large amount of pus, or in other cases, one would find the large mass remaining high with only slight evidence of pointing toward the vagina. Posterior colpotomy in these cases presented difficulties in technic, time consumption, inability of finding the pus,

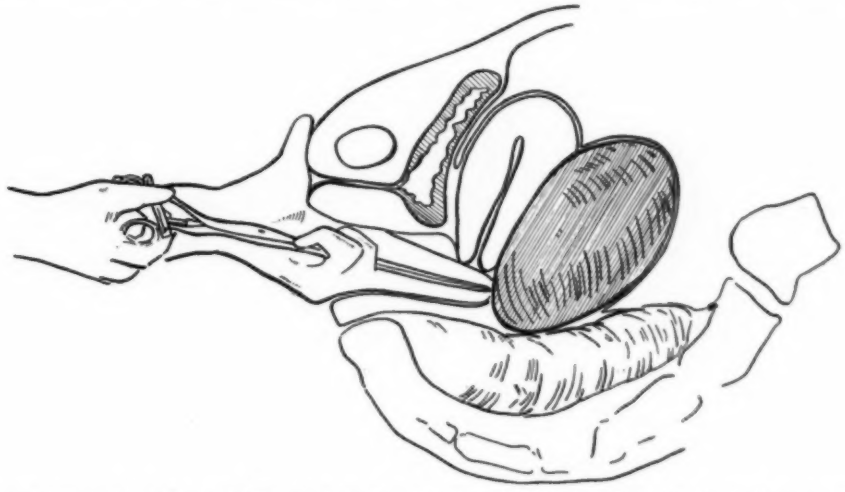

Fig. 2. Instrument passed along middle and index finger of left hand until it strikes posterior cul-de-sac behind cervix.

When an abscess is formed, one establishes the free escape of pus with a bistouri. In order to establish drainage, one must be sure that the abscess points in the direction of the possibility of perforating the adjacent viscera. Hence the procedure, although a good one, has gradually fallen into disrepute.

In 1924, L. Fraenkel[14] of Breslau, described an instrument for the rapid and accurate drainage of pelvic exudates.

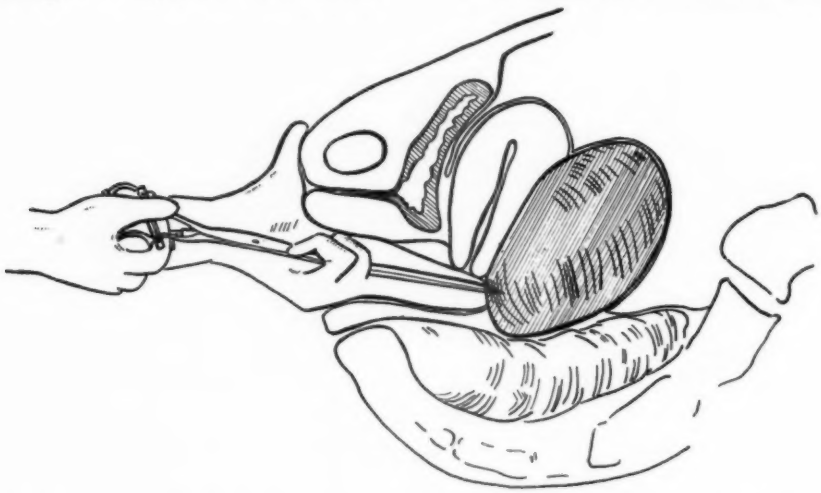

Fig. 3. Trochar pushed forward by index finger of right hand until instrument is fixed in place.

This instrument is built on the plan of a glove stretcher dilator and a trocar cannula (Fig. 1). This principle is not entirely new as Nonat in 1860 described an instrument which he called a trocar-lanceola. His instrument combined the knife with the trocar. M. Chaput used an instrument called a pince-trocar in a straight and curved model. The characteristics of his instrument were that its blades were sharp, to puncture the mass, and on spreading the blades apart, one would widen the incision.

The Fraenkel instrument is an excellent combination of the trocar-cannula and spreading blade principles.

The technic of posterior colpotomy with this instrument is easily mastered and the entire procedure can be carried out under the lightest anesthesia. The index and middle fingers of the left hand are introduced into the vagina until they reach the posterior lip of the cervix. The instrument is passed along these two fingers with the trocar inside the cannula (Fig. 2).

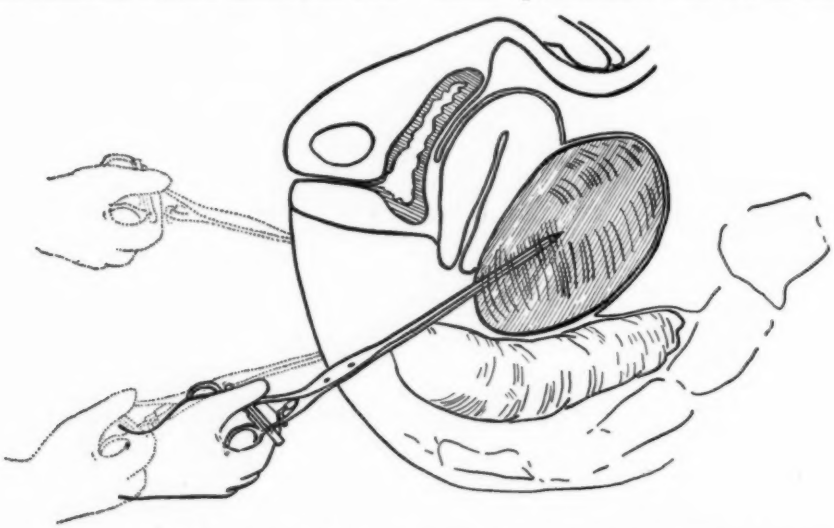

Fig. 4. Handle of instrument now depressed as far as possible and instrument pushed forward towards center of mass, using hand on abdomen as guide. Trochar is withdrawn to see if pus flows through cannula; if pus is obtained handle is then closed, thus opening blades and tearing hole in cul-de-sac.

When the end of the instrument impinges against the posterior cul-de-sac behind the cervix, the trocar is pushed forward fixing

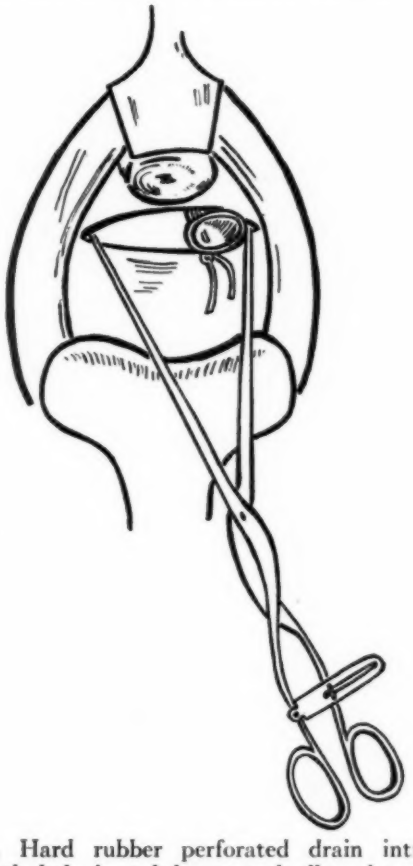

Fig. 5. Hard rubber perforated drain introduced through hole in cul-de-sac and allowed to remain there.

the instrument in place (Fig. 3). The left hand is then withdrawn from the vagina and placed upon the abdominal wall outlining the inflammatory mass. The handle of the instrument is now depressed as far as possible and the instrument pointed toward the center of the mass. It is then pushed into the mass for a distance of about 4 cm. (Fig. 4) or until a loss of resistance is felt. One then knows that the abscess cavity has been entered. The trocar is removed and the contents of the inflammatory mass allowed to escape. A culture and smear are both made from the fluid at this time. The lock on the handle of the instrument is now released and the handle of the instrument closed, thus opening the blades. This bluntly tears through the tissue, pushing vessels and ureters aside, and thus avoiding the severe bleeding sometimes encountered with the other method of

Fig. 6. Three sizes of hard rubber drainage tube showing arrangement of perforation.

colpotomy. When the instrument is opened to its fullest extent, it is again locked with the lockscrew and the hard rubber perforated drain is introduced through the hole in the cul-de-sac (Fig. 5). The drain is inserted up to its slightly projecting shoulder. Through one of the perforations in this drain a cord is tied to facilitate its removal. The Fraenkel instrument is then withdrawn and one strip of iodoform gauze is placed in the vagina. The gauze is removed the next day.

The after-treatment of the infection consists in helping the body resorb the exudate and in allowing the exudate to be discharged through the drainage tubes. The associated use of diathermy, hot air treatment, foreign protein therapy and usual supportive measures has given us excellent results.

As soon as the patient's temperature becomes normal, she is allowed out of bed. The drainage tube is never removed before the tenth day; occasionally if the exudate is still present but the temperature is normal, we allow the patient to go home and return to the out-patient department for treatment, removing the tube as late as the sixth week. The advantages of the Fraenkel posterior colpotomy lies in its extraordinary simplicity, the rapidity with which it can be performed and the shortness and lightness of the anesthesia. Fraenkel advocates the use of a hard rubber conical tube because "the concentric tension on the vault of the vagina which, as is known, tends to heal and bring together the cut edges, rapidly

brings compression and clogging of a soft rubber drain. This very centripetal tension of the fornix vaginae holds the hard rubber drainage tube in place." Should another abscess develop on the other side, one may wait a few days and puncture it the same way. If the tube becomes clogged, it is readily removed under vision and another one is inserted.

With the use of this instrument it is not necessary to wait until a tense cystic swelling can be felt in the cul-de-sac. The etiology and location of the inflammatory mass is not of tremendous importance. The important factor is that the mass be encapsulated. In parametric and perimetric infections, we find almost ideal indications for this colpotomy; even in the hard infiltrated masses where no pus is found the colpotomy relieves tension and allows the infectious material to escape. In high intraligamentous infections, where the mass presents itself on the anterior abdominal wall as well as in the pelvis, the colpotomy with extraperitoneal drainage from above will usually clear up the infection.

In those cases where a laparotomy has been performed and a large pyosalpinx, or markedly indurated and infiltrated parametrium is found the opening of which is distinctly contraindicated from above, puncture and drainage of this mass from below under direct vision have given very good results. In the past two years, we have performed this procedure twenty-one times. There have been no complications such as uncontrollable hemorrhage, perforation of a viscus or the development of a fistulous tract from the abscess.

CONCLUSIONS

1. With this method we often obtain a quick safe absorbtion of a pelvic exudate.
2. The mortality is nil.
3. The morbidity of patients with pelvic infection is markedly reduced.
4. Ovaries, tubes and uteri which owing to the infection would be scarified if a laparotomy were performed can be saved and the patient made symptom free.
5. It can make cases which at first seem inoperable, operable and may bring about the conservation of the pelvic organs.

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THE TRAUMATIZED KIDNEY

MEASURING THE PERCENTAGE OF DISABILITY*

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AFTER a complete search of the literature for the past ten years I find that little or nothing has been done in the matter of checking up the after-effects of renal injury. It has seemed sufficient to most observers and urologists to be quite satisfied if a patient with a bleeding kidney from traumatism ceases to show hematuria in a fairly satisfactory length of time and is discharged without inquiring to any extent into the exact amount of damage accrued. The coming of industrial-accident boards and commissions with the universal compensation insurance at present in vogue, the damage suits attendant upon automobile and other accidents, not only will now create an incentive to the study of the after-effects of injuries to the kidney but will force upon the medical profession the necessity of being able to report the exact percentage of loss of function.

I have been able to find only 4 cases in the literature to date where the functional end-results of renal injury have been studied.

Shupe reports the case of a young man who fell 20 ft., hitting his back in the descent and landing on his feet on a hard pavement. He complained at once of renal colic and showed a slight tenderness in palpation of the left kidney. There was very little shock but considerable blood in the urine. His treatment consisted of rest in bed. In two days there was no macroscopical blood and in one week it had disappeared when examined by the microscope. Two weeks afterwards cystoscopy was done and both kidneys found to be functioning normally.

Wesson reports 3 cases, one an eight-year old boy, run over by an automobile. There was right iliac fracture and crushing of the second and fourth lumbar vertebrae. There was hematuria for a few days and, after two months had elapsed, a swelling in the right side developed. This was incised and a large amount of clear fluid containing urea was evacuated. Cystoscopy and functional tests at this time showed both kidneys to be normal.

A man, forty-one, developed a large swelling on the left side one month after being struck by a car rail at that point. The swelling was opened, and about a gallon of urine drained. At this time cystoscopy showed no urine from the left kidney. One month later, after healing of the sinus, the functional output revealed a 50 per cent lessening from the left kidney as compared with the right but neither casts nor evidence of infection.

A man, forty-five, fell 25 ft. and struck on the right side, breaking several ribs. Hematuria lasted for thirty-one days. At the end of this time cystoscopy and functional tests showed a 50 per cent reduction from the right kidney. Re-examination after one year by cystoscope and pyelogram revealed the upper calyx replaced by scar tissue, the surviving renal portion being entirely normal and functioning the same 50 per cent.

My own personal experience has to do with 12 cases.

CASE I. Male, aged thirty-three, with a history of injury to the right side, accompanied by pain and hematuria, four years previously. Patient now enters the hospital complaining of frequent attacks of soreness and pain in the right side. Cystoscopic and roentgenological examination reveals an intensive pyuria, an absolute loss of function of the right kidney, a large mass of irregular shadows in the right renal area, and by pyelogram an immense calculus pyonephrosis. That this condition is a result of the kidney rupture is apparent, because at the time of injury a roentgenogram showed no shadow in the right renal area.

CASE II. Female, aged thirty, referred by an attorney for examination relative to a suit for damages. Patient received an injury to the right kidney two years ago, characterized

*Submitted for publication February 8, 1930.

by hematuria lasting three weeks with pain and tenderness. These symptoms were followed by chills and fever and a diagnosis was made of pyelitis of the right kidney on account of the symptoms and the presence of pus in the urine. Patient still complains of pain and pyuria and has started suit for damages. My cystoscopic and roentgenological examination revealed the urine from the bladder and both kidneys to be free from pus; the renal functional test showed an equal and normal output from both kidneys and the pyelogram revealed a normal right renal pelvis.

Case III. Male, aged thirty-eight, referred by attorney for examination. One and a half years ago, after unusual muscular exertion, he complained of extreme pain in the right side and hematuria for four days. At this time he states that he has severe pain in the back, becoming agonizing when bending over. He walks in a stiffened upright position when conscious of observation. When observed without his knowledge, he seems to bend fairly freely. Cystoscopic and roentgenological examination revealed a double or fused kidney on the opposite side, an equal and normal functional output from both kidneys, and pyelographic study fails to show any pathological condition of his right kidney.

Case IV. Boy, aged fourteen, struck by automobile, evident injury to the left side and kidney, accompanied by fairly severe hematuria which gradually let up but continued for twenty-one days. Two months afterwards examination revealed the urine microscopically free of blood or pus and by cystoscope both the kidneys were found to be functioning equally and normally.

Case V. Male, aged twenty-eight, painter, fell 20 ft. from scaffold, striking on his back; complained of severe pain in the right side, and at first urination noticed a considerable amount of blood. A fracture of the twelfth rib is noted on the roentgenogram. This patient was extremely restless and difficult to keep in bed. The physical signs and pain were probably aggravated by the rib fracture. No tumor could be palpated and the patient showed no signs of shock, and although the hematuria was rather severe, vigilant expectant treatment was decided upon. No complication occurred and the patient finally made a good recovery. Four months later he was given a routine examination which revealed a perfect and equal function from both kidneys.

Case VI. Female, aged thirty-two, entered the hospital with a history of having been thrown down and kicked in the back by her husband two months previously. At this time she had hematuria and severe pain in the left kidney region. Under rest in bed the hematuria subsided, but the pain continued in a lessened amount; in the last three weeks it has increased again and tenderness is more marked than before. At examination there is marked tenderness in the left costovertebral angle and flank. Cystoscopic and roentgenological study shows a non-functioning calculous pyonephrosis of the left kidney, which no doubt antedated the injury. Incision of the loin revealed a good sized perinephritic abscess which was drained, and, on account of the rather poor condition of the patient, the nephrectomy was postponed for three weeks.

Case VII. Female, aged twenty-eight, eight months ago slipped and fell coming out of a building where she was employed. Complained immediately of severe pain on the left side. She was removed to her home in a taxi, and at the second urination considerable blood was noted in the urine. She was removed to a hospital where cystoscopic examination was performed. I am unable to state with certainty whether catheterization of the ureters was likewise accomplished. After expectant treatment for twelve days, the blood entirely disappeared. The side, however, remained sore for a considerable length of time, and two months after the injury she was referred to me for examination. Cystoscopy revealed a normal urine, a normal functional output from the injured side, and the ureteropyelogram delineates a normal ureter and renal pelvis.

Case VIII. Female, thirty-four, struck by automobile and slightly injured. Complained shortly afterward of pain in the right side and back accompanied by vomiting and malaise. The urine was only slightly reddened after the injury, and blood was not discovered until three days later when her family physician had the urine analyzed. The bleeding was scarcely sufficient to be noticed macroscopically, but the urine continued more or less turbid for two weeks, when a chill accompanied by a rise in temperature to 103°F., and increased pain developed. Under hot applications and

urinary antiseptics, the symptoms subsided in a few days, but the urine at times continued cloudy. A suit for damages having been started, the patient was referred for examination seven weeks after the accident, and at that time the urine contained a considerable number of pus cells, which upon ureteral catheterization were found to proceed from the right renal pelvis. The renal functional test at this time was normal. The pelvis was thoroughly irrigated and an antiseptic instilled. One week later the urine from the bladder and right renal pelvis was found to be free from pus, the renal function again normal, and the ureteropyelogram showing a normal renal pelvis with a slightly dilated ureter. A careful study of the patient's antecedent history reveals that she had been treated for pyelitis of the right kidney during two pregnancies.

In 4 other cases of the usual type of injury, followed by hematuria and evidence of rupture of the kidney, I have been able to make cystoscopic and functional study only going over a period of from one to four years subsequent to the injury. In all four of these there is absolutely no evidence of any deleterious effect upon the functional capacity of the kidney.

SUMMARY

After careful perusal of the literature of the past ten years as published by Wesson, Fox, Pisani, Delzell and Harrah, Campbell, Bailey, Graves and Casper, Jefferson, Klingensmith, Marshall, Reese, Shupe, and Young, and from my own experience, I believe that the result of traumatism to the kidney, as viewed in terms of the percentage loss of functional disability, depends entirely upon the amount and type of destruction of the organ and the presence or absence of complications, particularly infection. My study of those patients injured and not subjected to nephrectomy, who have recovered without the intervention of infection, would surely prove that by far the vast number of ordinary cases of rupture of the kidney recover without any appreciable diminution in the functional ability of the injured kidney. If infection takes place and it is relegated to the renal pelvis alone, there may or may not be alteration in the kidney function. Such cases should be easily curable under the attention of a competent urologist by renal lavage and urinary antisepsis. If infection includes also the parenchyma, then serious results are bound to follow, as pyelonephritis, pyonephrosis, and possible renal calculus are almost sure to develop. As has heretofore been noted, the presence of preexisting renal disease must be taken into consideration in all kidney injuries and it will at times become a difficult problem to satisfy oneself in this matter.

In all events, the exact condition following traumatism to the kidney can be absolutely checked up by the present-day methods of cystoscopic and roentgenological study.

MEDICAL ASPECTS OF THE TREATMENT OF BENIGN PROSTATIC HYPERTROPHY*

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THE medical aspects of the treatment of benign hypertrophy of the prostate are summed in the fact that this condition produces obstruction to the urinary tract in elderly men; 85 per cent of the cases demanding operative relief are over sixty years of age. I propose to deal particularly with two phases of the problem: with the evaluation of the resultant functional renal impairment and its treatment, and with some of the complicating factors due to senescent changes in the cardiovascular system.

It seems scarcely necessary to say here that a careful and complete physical examination of the patient should constitute one of the first steps in the treatment of this disease. Such may disclose complicating or coexisting abnormalities (nephritis or heart disease) which must be dealt with as promptly as the urinary obstruction itself.

With hypertrophy of the prostate obstruction of the urinary tract distal to the bladder gradually develops. The anatomical changes which result are well-known. It is necessary here only to draw attention to the fact that the effects of back pressure are bilateral as far as the ureters and kidneys are concerned. The bladder wall hypertrophies, and trabeculation becomes evident; in many cases diverticula appear. The uterers are dilated irregularly; the renal pelves are enlarged and encroach upon the parenchyma of the kidneys. Of the local changes in the kidney so much may be said: in the absence of infection the microscopic alteration in the tubules are far more pronounced than are those in the glomeruli.

The impairment of renal function is generally assumed to be the result of three factors:

1. Back pressure in the urinary tract, even in the early stages, before atrophy of the kidney parenchyma occurs, its powers of elimination are seriously affected.
2. Infection, leading to cystitis, pyelitis, and sometimes pyelonephritis, and
3. Primary renal disease, the result of previous nephritis or of vascular changes in the kidney.

To these should be added a fourth: the functional capacity of the kidney is diminished by any appreciable degree of circulatory insufficiency.

The extent of functional damage may be evaluated by chemical analysis of the blood (it is customary to measure the blood urea) and by the ability of the kidney to eliminate phenolsulphonephthalein. Of the greatest diagnostic and prognostic significance are the successive estimations of the blood urea and of the phthalein excretion after free urinary drainage has been established. Following this, and after the administration of adequate quantities of fluid, the blood urea usually falls and the output of phthalein increases. If, after these procedures, renal function is not improved or restored it must be assumed that irreparable structural damage to the kidney has taken place. Careful physical examination may have revealed the cause of some part of this. Thus, in some cases, the presence of palpably thickened peripheral arteries and narrowed, tortuous arteries in the fundi oculorum, often with patches of exudate in the retinae, indicate widespread vascular disease. It is scarcely to be expected that prostatectomy will cure this state of affairs, although the relief of urinary obstruction and the elimination of infection in the urinary tract, may prolong the patient's life.

* From the Medical Clinic and the James Buchanan Brady Urological Institute, Johns Hopkins Hospital, Baltimore. Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, April 16, 1930.

Consider briefly the records of two typical cases:

The first, a man of sixty-five, was admitted with complete urinary retention. The blood urea at that time was 100 mg. per cent and the output of phthalein following catheterization was 11 per cent, with an appearance time of fifty minutes. A retention catheter was inserted, and fluid was administered up to 4000 c.c. daily. Four weeks later the blood urea had fallen to normal (30 mg.) and the phthalein excretion had risen to 30 per cent with an appearance time of only eleven minutes. Normal renal function was by no means restored but considerable improvement had occurred.

In the second case, a man of fifty, there was but slight urinary obstruction; the blood urea at the time of admission was 80 mg. per cent and the phthalein excretion 15 per cent. The phthalein appearance time remained at eight minutes for all the observations. The patient had pronounced hypertension, blood pressure 250/140, thickened tortuous arteries, and cardiac hypertrophy. His eye-grounds showed conspicuous papilledema, hemorrhages and huge patches of exudate. Despite the institution of free urinary drainage adequate renal function was never established. The development of myocardial insufficiency, although the patient was digitalized, handicapped the administration of fluid. Two weeks later the patient died. In the meantime the blood urea had risen to 300 mg. per cent and the phthalein had decreased to a mere trace.

In the first case the functional incapacity of the kidneys was due, for the most part, to the obstruction of the urinary tract. In the second it was incidental to generalized vascular disease. Of particular interest is the fact that, although the total excretion of phthalein in the second case was lower than in the first, the dye made its appearance in the urine within the normal time after injection.

To consider only one phase of the effect of age in rendering more difficult the problem of prostatic hypertrophy, any operative procedure upon a patient in the seventh decade of life or thereafter is undertaken in the face of a diminished circulatory reserve.

In the younger individual, in the absence of cardiovascular disease, the circulation, with the aid of its compensatory mechanism, is adequate to meet the most varied demands. With advancing age, however, coincident with degenerative changes in the blood vessels and myocardium, its functional reserve gradually decreases. The arteries become more or less sclerosed; irregular plaques are formed in the intima and may involve the medial coat. Diminution in the caliber of the vessels and impairment of the elasticity of their walls result. The changes in the myocardium are somewhat more varied. In the individual fibers histological alterations, pigmentation, occur which make it possible to distinguish an old fiber from a young one. Furthermore the myocardium may show hypertrophy or fibrosis. The first is the normal response to the increased work required to maintain an adequate blood supply through narrowed, inelastic arteries. Unless the valves are damaged or the resistance in the pulmonary circulation is increased, this enlargement is confined to the left ventricle. Secondarily the aorta dilates, chiefly in the region of its base and arch. The second change, myofibrosis, is due to one or both of two factors: First the normal increase in interstitial connective tissue and wasting of muscle fibers, which occur with age, secondly to localized or diffuse muscular degeneration and its replacement by fibrous tissue incident to impairment of the blood supply to the myocardium. The latter is augmented by any sclerotic narrowing of the coronary arteries either at their origin in the aorta or along their course.

Whatever may be the anatomical results of such changes the inevitable physiological effect is an impairment of the capacity of the heart to meet increased demands. The heart's efficiency depends upon the integrity of its metabolism; this presupposes an adequate blood supply to the myocardium in proportion to the load placed upon it. The circulatory requirements under normal conditions can, in the majority of cases, be supplied, but

unusual demands (over-exertion, fever, hypertension etc.) cannot be met without some evidence of circulatory insufficiency.

Physical examination of the senile heart frequently presents the following clinical picture: The impulse is often obscured by overlying emphysematous lung; when visible or palpable it is usually displaced somewhat to the left. The relative cardiac dullness is enlarged, particularly to the left and downward, and the retrosternal dullness, in the first and second interspaces, is widened, corresponding to the dilated aorta. The heart sounds at the apex are distant and feeble, and are often accompanied by a blowing murmur (functional mitral insufficiency). Over the base the aortic second sound may be sharp but, in the absence of hypertension, is not greatly accentuated. More characteristic is a systolic murmur in the second right interspace, sometimes transmitted upward. This is produced, supposedly, either by arteriosclerotic stiffening of the aortic cusps or by the relative disproportion in the diameter of the normal aortic orifice and the dilated arch. The cardiac rhythm may be normal but is often interrupted by ventricular extrasystoles. Rarely auricular fibrillation may be present.

In the absence of myocardial insufficiency such patients present few symptoms referable to the heart. They are conscious of limitation of their capacity for physical work, and occasionally complain of palpitation; this latter symptom is sometimes lacking even in cases showing numerous extrasystoles. As the circulation becomes overtaxed, however, more obvious symptoms develop: fatigue, breathlessness while talking, dyspnea on slight exertion and orthopnea. Finally a small proportion of these patients complain of typical anginal pain. The physical signs of myocardial insufficiency may be present in varying degrees. These include evidence of pulmonary edema, râles at the lung bases, hepatic enlargement, and edema of the extremities.

It has become increasingly apparent that the frequency and severity of postoperative complications may be materially lessened by adequate preoperative preparation.

Rest is a most important measure. Frequency of urination particularly at night may have so interfered with sleep that the patient is well-nigh exhausted. This may be relieved by catheter drainage. Sedatives, luminal or even opiates, should be prescribed as required.

Diet is often a perplexing problem. In the acute stages, with nitrogen retention, milk is borne best, (800 to 1000 c.c. daily). Subsequently a more complete diet may be administered. In cases with edema salt should be restricted (1.0 gm. or less). Even in the presence of nitrogen retention it is useless to reduce the protein intake below that amount necessary to maintain the patient in nitrogen equilibrium (50 to 60 gm. per day).

Fluids. The impairment of renal function due to obstruction of the urinary tract appears to interfere less with the excretion of water than with that of nitrogenous waste products. To the end that adequate nitrogen excretion may be accomplished as well as to combat the infection in the urinary tract it is often necessary to administer large quantities of fluid to these patients. This constitutes, sometimes, a very vexing problem. If the patient is not already nauseated forcing fluids by mouth may make him so. If signs of circulatory insufficiency are present too large a fluid intake may only make a bad matter worse.

It seems most advisable to determine first whether any degree of circulatory insufficiency exists. If such is the case it is unwise to force fluid at once but better to increase the fluid intake gradually after one or two days' rest and digitalization. In general, too, the administration of fluids by the intravenous route, in elderly patients, is to be undertaken with caution.

When this appears imperative the following method has proved useful. It is similar to that suggested several years ago by Dr. Matas. A silver cannula is inserted into a superficial vein, usually on the foot,

and is tied in place. Through this normal salt solution is injected continuously, regulated by a drop device quite similar to the Murphy drip so that the patient receives not more than 100 to 200 c.c. per hour. In this fashion large amounts of fluid may be given over a period of days and so slowly that the circulation is not embarrassed thereby.

Digitalis. As has been so often stated the indication for digitalis is myocardial insufficiency; the routine administration of this drug is not only useless but unwise. It should, however, be given to any patient showing signs of congestive failure, and in adequate quantity, i.e., 1.5 gm. standard leaves per 100 lb. body weight, or an equivalent amount of some standardized preparation. This is best administered in divided doses over a period of forty-eight hours or more, except in cases of acute failure to which it may be given more rapidly. After the maximum therapeutic effect has been obtained digitalis should be continued at 0.1 to 0.2 gm. daily to replace the amount normally excreted. If the patient has received this drug prior to admission digitalis must be administered in smaller doses and a longer period allowed for digitalization in order to avoid intoxication. The development of acute dilatation to which we shall refer presently calls for an increase in digitalis dosage, or, if the patient has not been fully digitalized previously, for strophanthin (0.5 to 1.0 mg. intramuscularly). In cases showing numerous ventricular extrasystoles the combination of digitalis with dionine may often restore the normal rhythm more quickly than does digitalis alone. This is given in capsules (Fol. digitalis 0.1 gm., dionine 0.006 gm.) and continued up to the therapeutic maximum for digitalis.

Operation. The circulatory strain resulting directly from an operation is due to one or both of two factors. The first is reflected in a reduction of the vital capacity. The normal vital capacity falls steadily during the sixth and seventh decades of life and thereafter, until at eighty it is but 45 to 50 per cent that of the normal at thirty years of age. Following an operation, particularly an abdominal operation, this is often still further reduced. The chief circulatory strain may be due to the second of these factors, i.e. the anesthetic. Here the choice is often a nice one. Aside from increasing the frequency of postoperative pulmonary complications, general anesthesia, ether or nitrous oxide, is attended by a degree of anoxemia and by secondary circulatory changes which must be regarded as dangerous in elderly patients. Herein lies the advantage of caudal or of spinal anesthesia. Except in occasional, very apprehensive, patients with symptoms of angina pectoris we have used caudal or epidural anesthesia, injecting 20 c.c. of a 3 per cent solution of procaine into the sacral hiatus. Morphia (16 mg.) is given the night before operation to insure rest and again just before the patient is removed to the operating room.

Postoperative Treatment. Digitalis should be continued after operation in doses just sufficient to maintain the therapeutic effect (0.1 to at most 0.2 gm. daily). During the first two postoperative days this is usually best administered by injection, digifoline 1 to 2 c.c. The interference with respiration caused by abdominal distension may sometimes contribute to circulatory failure. This should, therefore, be prevented as far as possible, and vigorously combated as soon as it appears. Certain drugs, and particularly morphine, contribute to distension by relaxing the intestine. Schlesinger's solution and pantopon seem less likely to foster distention and at the same time are efficient sedatives. After prostatectomy any treatment per rectum, enema or passage of rectal tube, is to be avoided on account of danger of embolism. Turpentine stupes, pituitrin or eserine are usually effective in dispelling distension.

One of the most alarming postoperative complications is acute cardiac dilatation. In this condition the myocardium becomes suddenly unable to accomplish an adequate

systolic discharge. Symptoms and signs of circulatory failure rapidly develop, the patient becomes cyanotic, the pulse rapid and thready, the blood pressure falls, the pulse pressure is reduced, pulmonary râles become evident and the liver is engorged. The heart is often demonstrably enlarged, its sounds are feeble, and a gallop rhythm is frequently audible. The cause of dilatation is not always immediately evident. It is theoretically due to an increase in the myocardial load beyond the optimum limit in proportion to its oxygen supply.

The most common causes of this complication are pulmonary embolism and coronary occlusion. The abrupt blocking of a greater or lesser portion of the pulmonary circulation suddenly increases the resistance to the output of the right ventricle. The added anoxemia contributes to the failure of the myocardium. Coronary occlusion, by depriving a portion of the myocardium of its arterial supply, may bring about acute and alarming symptoms of dilatation. If the occlusion involves a large vessel death is the inevitable result. If, however, it occupies one of the terminal branches, compensation may later be restored. Minor coronary occlusion may be attended by symptoms and signs of cardiac dilatation and by moderate fever and leucocytosis.

The patient with acute cardiac dilatation is often conscious, restless and extremely apprehensive. A sedative, usually morphia, should, therefore, be given at once. If the patient has not been previously digitalized this should be accomplished as rapidly as possible, best of all with strophanthin. The fall in blood pressure observed in such cases is due less to peripheral vascular relaxation than to the diminished systolic output of the heart. The administration of adrenalin in an attempt to raise the blood pressure by constricting the peripheral vascular bed may only increase the load upon an already overburdened ventricle. The use of caffeine (0.2 to 0.3 gm.) to stimulate systolic contraction of the myocardium is rather more logical.

When is the patient to be considered ready for operation? This must obviously be decided for each case individually and by the urologist and internist in consultation. More important than the patient's condition upon admission is the degree and rate of his improvement under treatment. As stated previously following the establishment of free urinary drainage and the administration of adequate amounts of fluid, the blood urea usually falls and the phthalein excretion rises. It is well to postpone operation as long as this improvement increases. And yet, in many cases, after a temporary improvement the infection in the urinary tract tends to increase with catheter drainage. To delay too long may, therefore, undo some of the good which has been done. From the point of view of the circulation the return of circulatory compensation should be awaited before operation is attempted. The problem consists in picking the point of maximum improvement for each case.

SUMMARY

The importance of a careful and complete physical examination of the patient suffering from benign hypertrophy of the prostate is emphasized. Generalized vascular disease may play no small part in the renal insufficiency so often encountered in these patients.

Many of the medical problems of hypertrophy of the prostate have to do with the anatomical and physiological changes which occur with advancing age. Among them the alteration in the cardiovascular system are most significant.

The preoperative and postoperative treatment of this condition is discussed from a medical standpoint.

The author desired to express his appreciation to Dr. Hugh H. Young for permission to follow these cases on his service, and to acknowledge with thanks the assistance of the staff of the Brady Urological Institute.

PROSTATIC NEOPLASMS

THEIR DIAGNOSIS BY NEEDLE PUNCTURE AND ASPIRATION*

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THE use of some form of needle or trocar for obtaining biopsy specimens is not new. Goeller in 1920 devised a trocar with a spiral cutting tip for securing tissue from the prostate.[1] Mixter some thirty years ago and more recently Ward,[2] Guthrie[3] and Forkner[4] have devised trocars and needles for special purposes admirably adapted for securing tissue from the various sites in which they were especially interested. The Goeller trocar or prostatic screw as it is sometimes called has been in use both at the Memorial Hospital and the New York Hospital since its invention. In our hands its use has proved difficult. It is successful in obtaining tissue from the prostate in about 30 per cent of the cases to which it is applied.[1] To keep the instrument sharp and serviceable requires the services of an expert mechanic. We have used the trocar and have also attempted several modifications with more or less the same results.

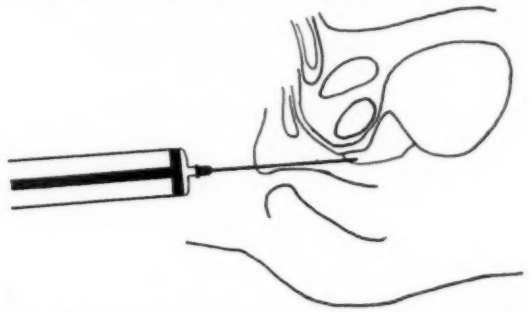

FIG. 1. Prostate in sagittal section showing needle and syringe in first position. Note point of needle just within capsule of prostate and syringe closed.

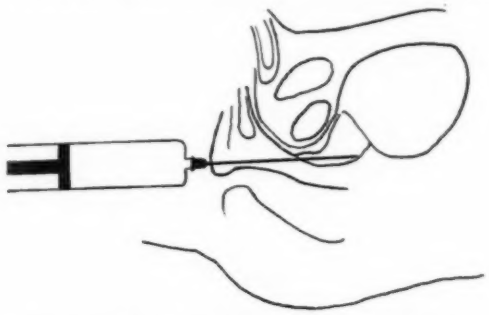

FIG. 2. Plunger of syringe held out as needle is advanced in straight line through suspected prostatic tissue.

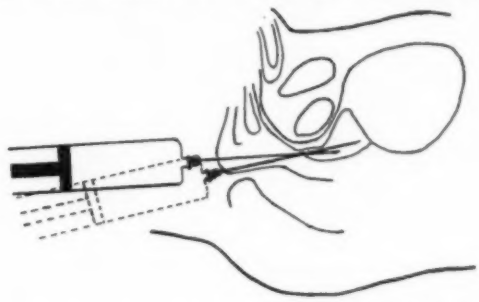

FIG. 3. Syringe in solid outline in third position, withdrawn about 0.5 to 1 cm. from position in Figure 2; suction maintained. In dotted outline, syringe is again advanced but at a different angle in order to cut off plug of tissue in bore of needle. Suction maintained.

Since 1926 Martin and Ellis[5] have employed aspiration with an ordinary 18 gauge needle attached to a record syringe. Martin has consistently applied the method to the general field of tumor diagnosis at the Memorial Hospital, while Ellis has developed a rapid technic for staining the specimens obtained. Their methods and experience in 65 cases are carefully detailed elsewhere and need no repitition here. At their suggestion we have applied the technic to the diagnosis of prostatic tumors with results so satisfactory that we have abandoned previous methods.

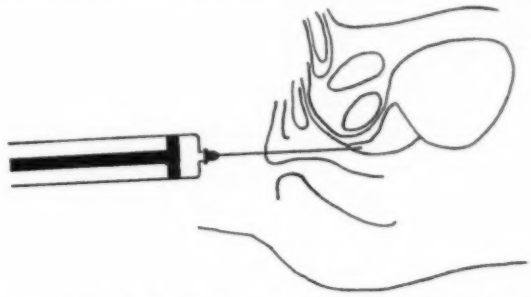

FIG. 4. Needle withdrawn to prostatic capsule. Syringe allowed to equalize slowly. Disconnect needle from syringe and quickly withdraw needle.

* From the Guggenheim Urological Fund, Memorial Hospital, New York. Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, May 21, 1930.

ADVANTAGES AND INDICATIONS

The advantages of such a method of dealing with prostatic lesions are numerous.

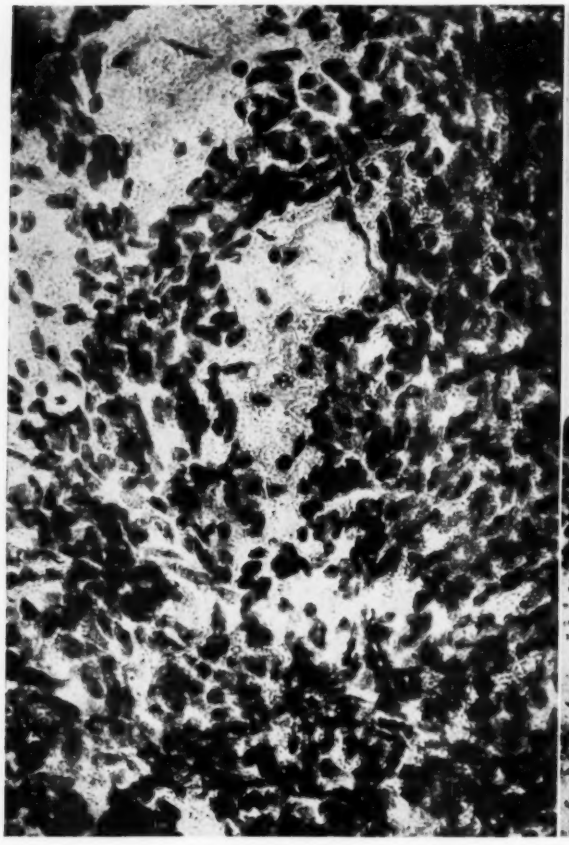

FIG. 5. Carcinoma of prostate. Aspiration biopsy from papillary malignant cyst.

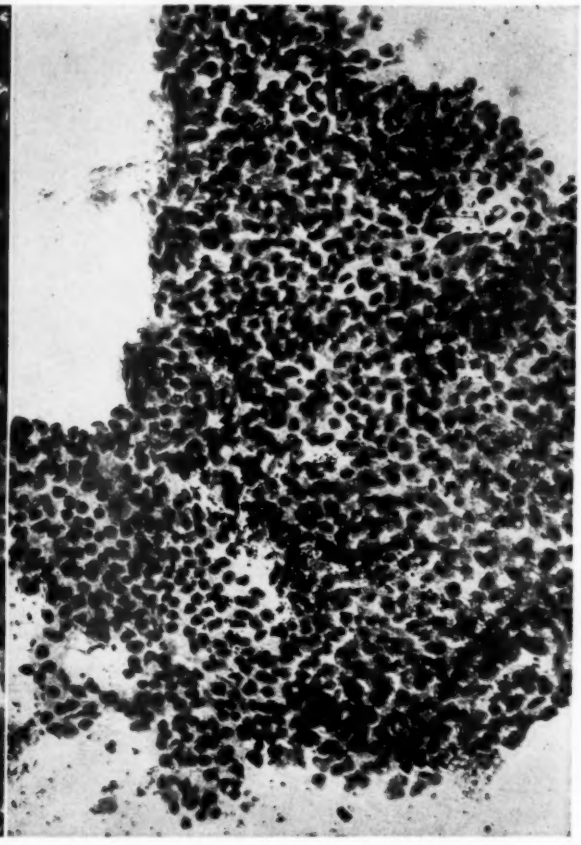

FIG. 6. Small cell carcinoma of prostate. Aspiration biopsy from solitary 2 cm. nodule. Early case.

The paraphernalia required are to be found in any hospital ward or clinic, readily available for impromptu use. The method is readily accepted by the patient without question, as little time and no extensive preparation are necessary. A much higher percentage of success is obtainable than with any previous method. In our hands 70 per cent of aspirations done succeeded in securing demonstrable tissue from the prostate for examination. Some failures will be occasioned by attempts to aspirate tissue from fibrotic myomata or adenomata and from neoplasms which have been heavily radiated. Some early failures were due to the use of a dull needle; we now sharpen the needle after each aspiration. Some more recent failures were due to the use of too short a needle in fat individuals with deep perinei. We find however that with strict adherence to the technic about to be described and observation of these details we become more uniformly successful. As to the dangers, Martin in five years' experience failed to find the clinical setting of any case essentially altered by the procedure. The trauma of a single puncture of the tumor wall by an 18 gauge needle may be considered negligible. As far as the prostate is concerned we have yet to see any ill effects in a series of very carefully observed cases. The chief field of usefulness of the procedure lies in the differential diagnosis of the early or doubtful case. We do not use the method where a larger specimen is to be obtained by surgical means. An interesting use of the procedure was made in 2 cases where a metastatic adenocarcinoma of the lung had been

proved by aspiration. Search for the primary growth in both instances was rewarded when aspiration showed adenocarcinoma in the prostate. Neither case had any symptom which would have called the attention of the patient to the prostate.

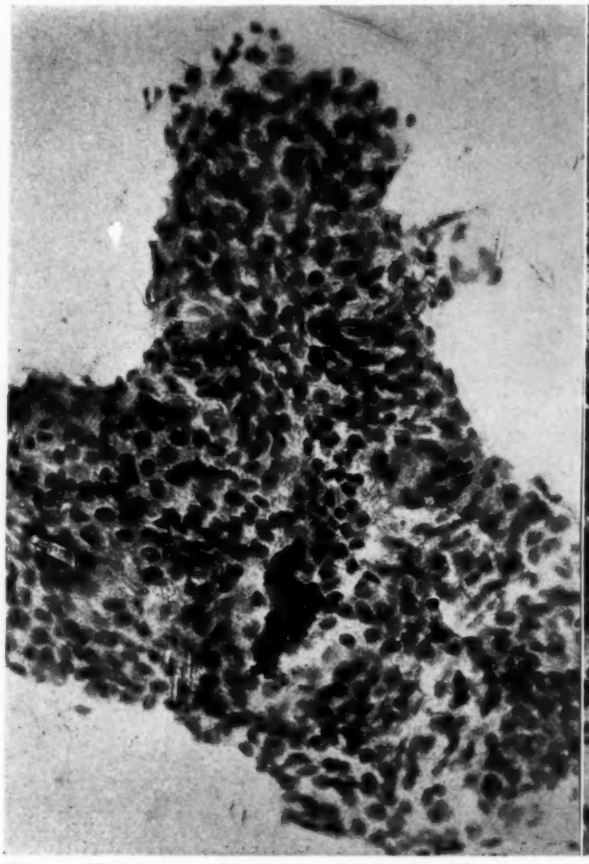

Fig. 7. Carcinoma of prostate. Aspiration biopsy.

Fig. 8. Carcinoma of prostate. Aspiration biopsy. From highly malignant growth.

Technic for Securing Specimen

The technic is as follows. The patient is placed in the lithotomy position on the table. The skin of the perineum is cleansed with alcohol. A single wheal of 1 per cent novocaine is raised on the skin just to one side of the median raphe about 1 in. anterior to the anus. This is followed by the infiltration of novocaine down to the prostatic capsule, guided by the index finger of the left hand placed in the rectum. The sterile Record syringe with an 18 gauge needle, 4 to 6 in. long, with long sharp bevel, is now taken in the right hand and inserted through the skin of the perineum avoiding the midline. With the index finger of the left hand in the rectum the needle is now carefully guided to the prostatic capsule avoiding both the rectal and urethral walls. Figure 1 shows the position of the needle in sagittal section at this juncture and also shows that the insertion up to this point has been accomplished with the plunger of the syringe closed. Now the plunger is drawn out as far as possible, creating a high vacuum in the system, as the needle is advanced in a straight line through the suspected nodule of tissue. This step is illustrated in Figure 2. The result of this step is that a small plug of tissue is sharply cut and drawn up into the needle. In order to cut off the tissue and successfully withdraw it, it is necessary to withdraw the needle about 0.5 to 1 cm. and then advance it again but at a different angle, as illustrated in Figure 3. During this manipulation it should be noted that negative pressure is continu-

ously maintained by holding the plunger of the syringe out. The needle is now withdrawn to the prostatic capsule, the pressure in the syringe slowly allowed to equalize (Fig. 4), and the syringe disconnected from the needle. The needle is now quickly withdrawn from the patient, the syringe filled with air and reconnected to the needle. The material in the needle is then slowly and carefully expressed on to a glass slide. It is then smeared with a second glass slide using firm but gentle pressure, as for a blood smear. Care should be taken to get the preparation thin enough for good visualization. Fixation is by gentle heat, after which the specimen is rapidly stained according to the technic described by Ellis and quoted later.

Fig. 9. Carcinoma of prostate. Four year recurrence. Slow growing tumor. Aspiration biopsy.

Fig. 10. Carcinoma of prostate. Aspiration biopsy, early case, small tumor confined to prostate.

PREPARATION OF THE SPECIMEN

Ellis describes two methods of preparation, one only being generally applicable to smears of tissue from the prostate. The other method is a paraffin embedding method to be utilized when larger amounts of tissue are obtained by the needle. This happens frequently with other tumors but rarely from the prostate. The immediate method is as follows: The fresh tissue fragment on the glass slide is smeared by very firm flat pressure by another glass slide drawn once across. The smeared slide is fixed by gentle heat until warm and dry and is then run through the following technic:

	Minutes
1. Alcohol (95 per cent)	1
2. Water	1
3. Hematoxylin	1
4. Water	1
5. Eosin	1
6. Alcohol (95 per cent)	½
7. Carbol-xylol	½
8. Mount with Canada balsam and cover glass.	

The times given may be varied slightly

and it is especially suggested that the staining of the specimen with hematoxylin be followed under the microscope.

cer. Gross morphological structure such as one would find in well cut paraffin sections is of course not to be found. Many

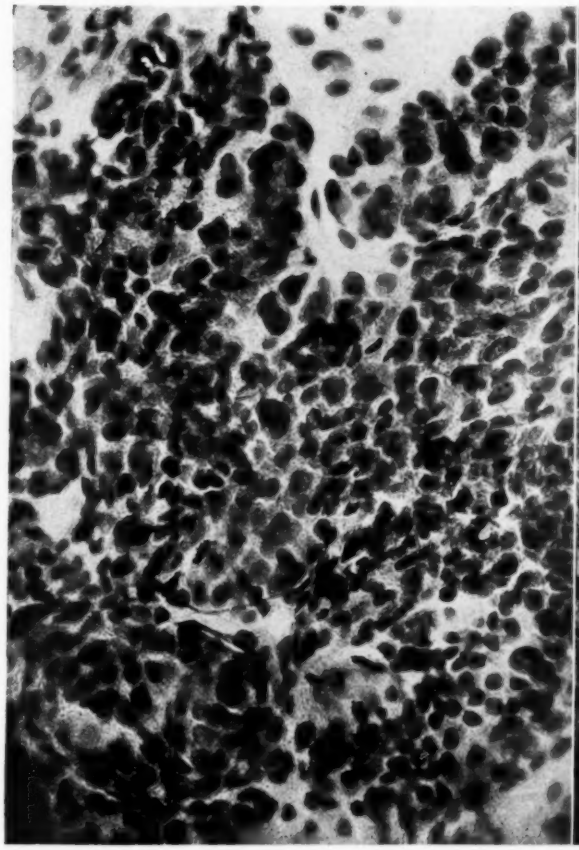

Fig. 11. Carcinoma of prostate. Aspiration biopsy.

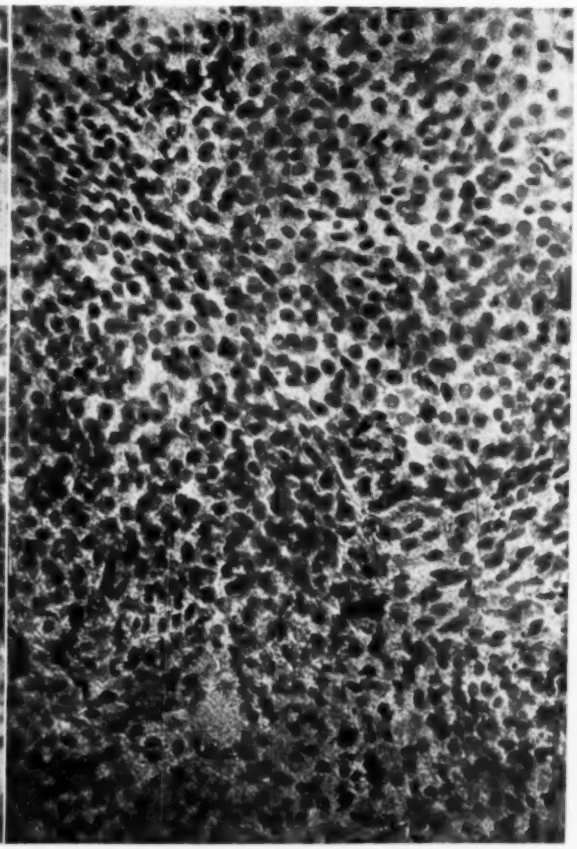

Fig. 12. Carcinoma of prostate. Aspiration biopsy. Small cell tumor.

INTERPRETATION OF RESULTS

The interpretation of the smear requires the services of a pathologist fully conversant with the field of tumor diagnosis. He must be associated with a clinician whose clinical interpretation of the facts of the case is accurate and reliable. In these smears careful search reveals small groups of cells having the definite cytological structure and morphological arrangement of cancer. While it would be very unwise to base a diagnosis on a single cell or very small group of cells yet it is easily possible to find enough material scattered throughout the smear, which when carefully studied can lead only to the definite conclusion that the tissue is or is not cancer. Gross morphological structure such as one would find in well cut paraffin sections is of course not to be found. Many pathologists would not be willing to hazard a diagnosis on such a small amount of tissue, but we know that with painstaking technic and a pathologist able and willing, a reliable diagnosis of cancer can be made on material obtained as described. Where such conditions do not prevail we would hesitate to recommend the procedure. Under properly organized conditions we feel that the method of biopsy described can be safely and routinely used in the diagnosis of prostatic neoplasms with a higher degree of success and satisfaction than with any method heretofore proposed.

The appended photomicrographs illustrate some of our results. [For references see Author's reprints.]

ACUTE APPENDICITIS*

STATISTICAL REVIEW OF 97 CASES

CURTISS GINN, M.D., F.A.C.S.

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THIS study was made with an especial view as to the present-day mortality in cases of acute appendicitis.

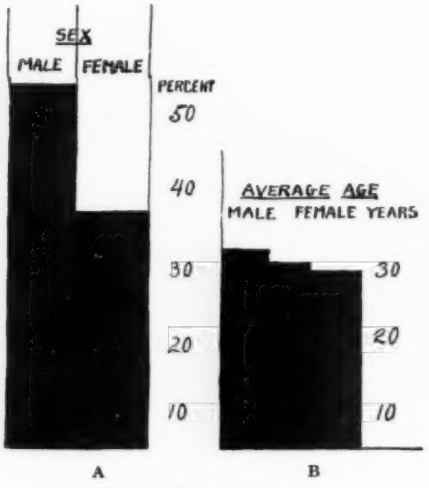


FIG. 1. Age and sex.

It has been my impression for several years, both from my reading and personal association, that the mortality is greater than it was a decade or two earlier.

While the number of cases is small and not large enough to make any very positive statement, on the whole it tends to substantiate the impression.

The study is based upon the cases of acute appendicitis operated upon at the St. Elizabeth Hospital during the year January 1, to December 31, 1929.

In order to arrive at an accurate survey, the following requisites were made essential for each case considered:

1. A complete and legible history. Physical examination and a provisional diagnosis of acute appendicitis made.

2. The surgeon's and pathologist's final diagnosis must agree, i.e. the case is one of undoubted acute appendicitis.

3. The case must have been uncomplicated by any other abdominal or pelvic pathology.

4. That at the time of survey the case must have been terminated and the chart closed under the heading of surgical cure or surgical death.

Only 97 cases could be found within the year which fully qualified under these four requisites and these cases form the basis of this report.

As regards the *environment:* The operating-room and service are well organized; the percentage of infections during the same year in other types of cases was equal to or even below the usual figures in most hospitals, the last survey of the infections in the hospital being 0.5 per cent for all cases operated upon.

In none of the cases that died was there any question of the responsibility resting upon the operating-room or the nursing personnel.

Seventeen different surgeons operated upon the 97 cases, as follows:

Eight of the operators were classed as men doing surgery exclusively.

Two were surgeons doing some general practice.

Six were primarily general practitioners doing some surgery.

Sex. Of the 97 patients:

58 or 59.8 per cent were males.
39 or 40.3 per cent were females.

Sonnenburg in 1000 cases (survey made in 1910) had a percentage of 67 males.

Deaver at about the same time, in 300 cases, found 61 per cent males.

Age. The average age of the males was 26.7 years. The average age of the females was 24.55 years. The average combined age was 25.62 years. This survey then is in general accordance with the statistics of larger groups of cases, in that it is primarily a disease of young

* Read before The Miami Valley Pathological Society, February 14, 1930.

adult life. The youngest patient was ten months of age. The oldest patient was seventy-nine years of age.

of the abdomen. In 80 per cent of the cases it was definitely located in the right lower quadrant.

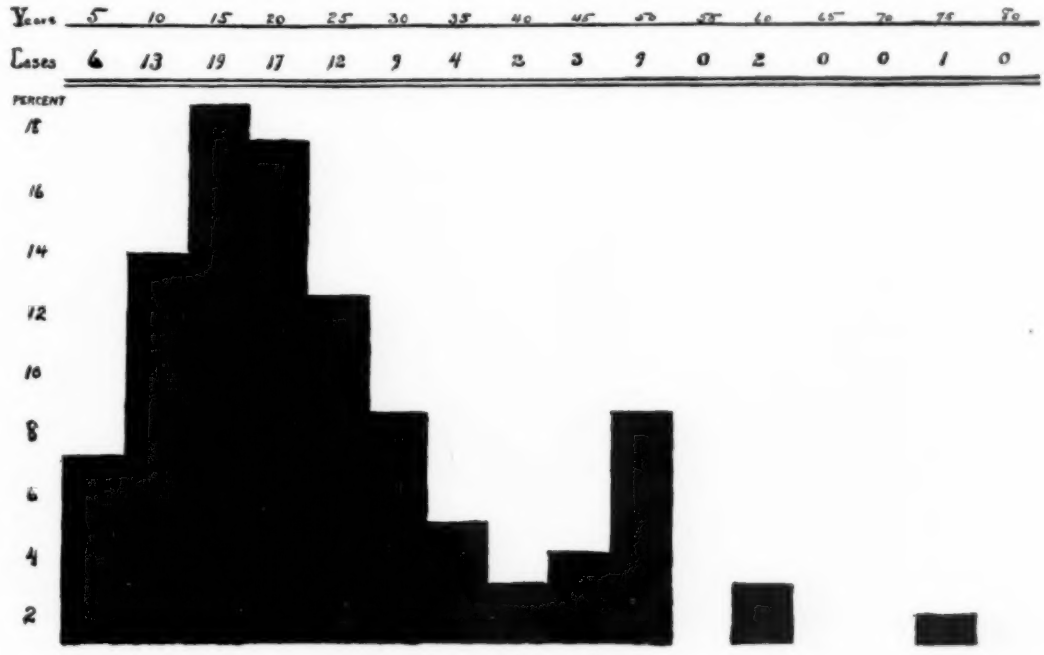


Fig. 1c. Age and sex.

Arranged in five-year periods we have the following:

Table 1

Years	Cases
1– 5	6
5–10	13
10–15	19
15–20	17
20–25	12
25–30	9
30–35	4
35–40	2
40–45	3
45–50	9
50–55	0
55–60	2
60–65	0
65–70	0
70–75	0
75–80	1

In this group there was no one recurring *etiological factor*, with the possibility of an immediate preceding tonsillitis in 3 of the cases under ten years of age.

Symptoms. The one symptom common to all was abdominal pain, generalized at first. This pain in a few hours, from four to six, became more intense in one quadrant

Nausea was the next most frequent symptom, present in 72 per cent of the cases.

Vomiting was present in 50 per cent, occurring only once or twice unless the case was complicated with some degree of peritonitis.

Constipation, from the beginning of the attack at least, was also the rule except in the rare instance where an appendicitis apparently followed hard upon the ingestion of a late hour meal of heavy food.

No case of twenty-four hour duration was entirely afebrile or had a temperature of more than 100⅖° F., except in children under ten years of age. After the first twenty-four hours the degree of temperature depended upon the local condition, the degree of tension of the pus, whether an extra-appendicular abscess was forming, and the degree of peritonitis existing.

I would like to say at this point that these cases with one exception were not recurrent cases.

Leucocytosis. Seventy of the 97 cases

had a white blood cell and differential count made after admission to the hospital and just prior to the operation.

Case No. 11848. Male, aged forty-nine, ill seventy-two hours. Perforated appendix and general peritonitis. Appendix removed, multiple Penrose drainage, in the hospital forty-two days. Re-opened on the twenty-fifth postoperative day, on account of persistent purulent drainage from abdominal sinus. Fecolith removed. Recovery.

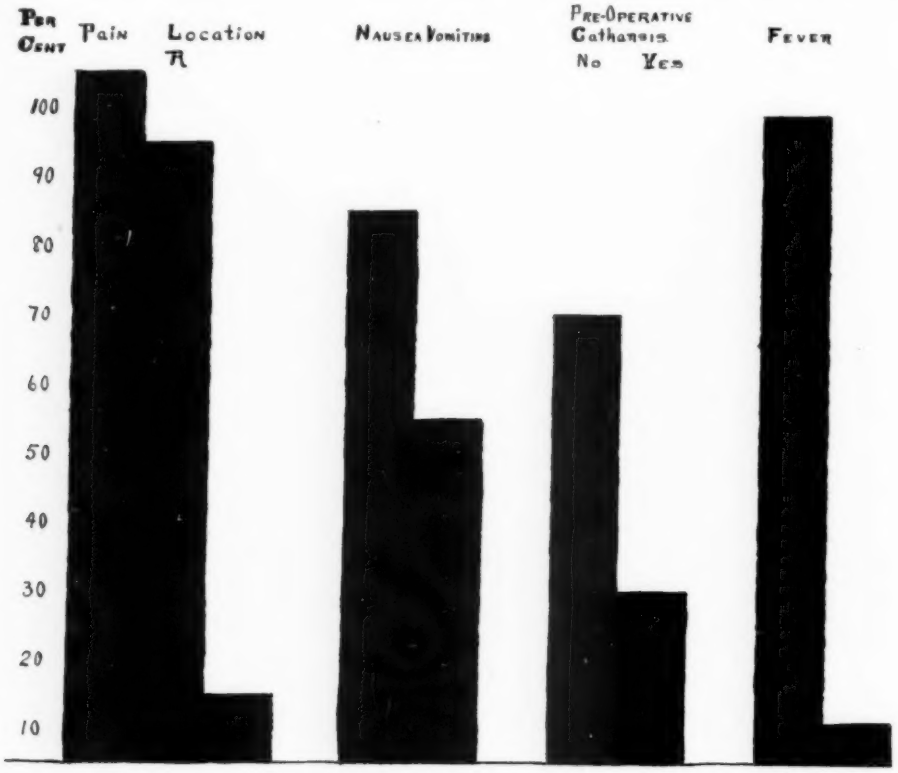


Fig. 2. Symptoms.

Table II

Cases	Leucocytes	Percentage
14	5,000–10,000	20
35	10,000–15,000	50
11	15,000–20,000	15.7
8	20,000–25,000	11.4
2	25,000–30,000	2.9

Table III

DIFFERENTIAL LEUCOCYTE COUNT

Cases	Polymorphonuclear (per cent)
2	50– 55
1	55– 60
3	60– 65
6	65– 70
5	70– 75
11	75– 80
19	80– 85
14	85– 90
9	90– 95
2	95–100

The two cases having between 50 and 55 per cent leucocytes were:

Case No. 4336. Female, aged ten, ill seventy-two hours. Gangrenous appendix, general peritonitis. Slough of appendix removed, Penrose drain to appendiceal stump and right renal fossa, tube stab drainage, midline, for pelvis. No complication. Hospital stay twenty-three days. Recovery.

One case had between 55–60 per cent:

Case No. 4336. Female, aged 7, ill fourteen days. Perforated appendix, general peritonitis. Hospital stay eight days. Died of Peritonitis. 6800 leuc., 58 per cent polys.

Three cases had between 60 and 65:

Case No. 4588. Male, aged one year, ill fourteen days. Perforated appendix, general

peritonitis. Appendix removed and tube drainage. Died three days postoperative of peritonitis. 9800 leuc., 60 per cent polys.

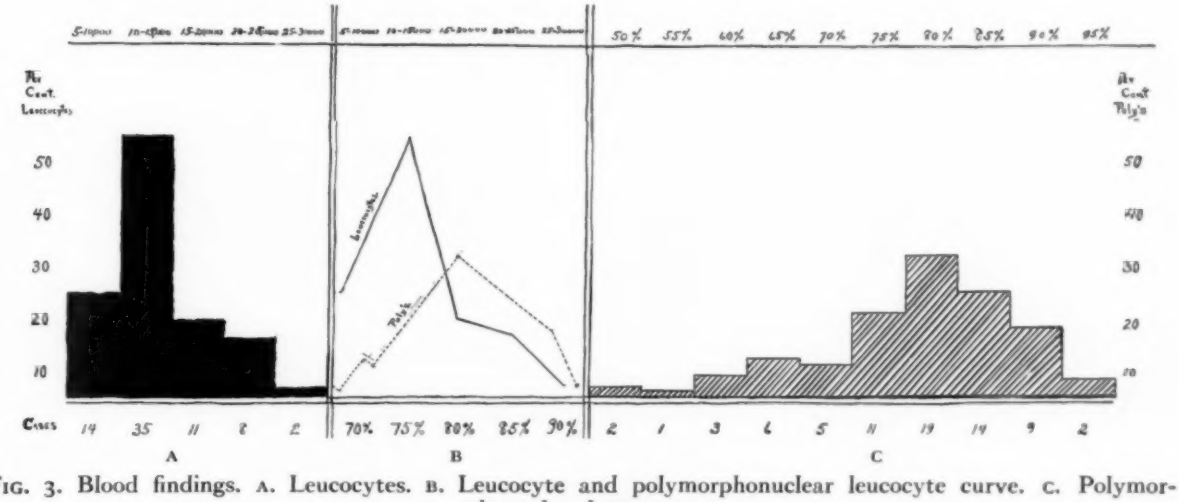


Fig. 3. Blood findings. A. Leucocytes. B. Leucocyte and polymorphonuclear leucocyte curve. C. Polymorphonuclear leucocytes.

Case No. 4525. Female, aged thirty, ill twelve days. Perforated appendix, general peritonitis. Appendix removed, tube drainage. Hospital stay fourteen days. Cured. 9000 leuc., 64 per cent polys.

Case No. 3476. Male, aged twelve years, ill seventy-two hours. Acute appendicitis, local peritonitis, confined to right iliac fossa. Appendix removed, Penrose drainage. Hospital stay twenty days. Recovery. 10,000 leuc., 58 per cent polys.

Twenty years ago J. B. Murphy wrote in his classic on Appendicitis:

The percentage of polynuclear cells is an indication of the severity of the infection, but not of the degree of destruction; a high percentage does not denote a bad prognosis, so long as the absolute number of white cells is correspondingly high. A fall in the absolute number of polynuclears with a coincident decrease of the total percentage of leucocytes shows a decline in the infection. A sudden fall in the absolute number of polymorphonuclears, with a very small decline or perhaps a slight percentage of white cells, is of unfavorable prognostic import. A very unfavorable sign is a sudden fall in the total number of leucocytes.

For many years, in general, surgeons have taken the views just expressed, but this past year two papers on this subject have been published. One by Eggers[2] states the degree of leucocytosis is not an indication of the severity or the extent of an atack. The simple establishment of a leucocytosis is enough to make a diagnosis of acute appendicitis. The same is true of a differential count.

Hellwig[3] in discussing the leucocyte count in 160 cases writes that the height of the leucocytosis bears no relationship to the severity of acute appendicitis. The white blood cell count does not run parallel to the different stages of the pathological process. The practice of not advising operation in acute appendicitis unless there is a marked increase in the number of leucocytes is dangerous. Appendectomy must not be undertaken nor deferred on the basis of the blood count alone. Leucocytosis is a minor symptom, inconstant and unreliable.

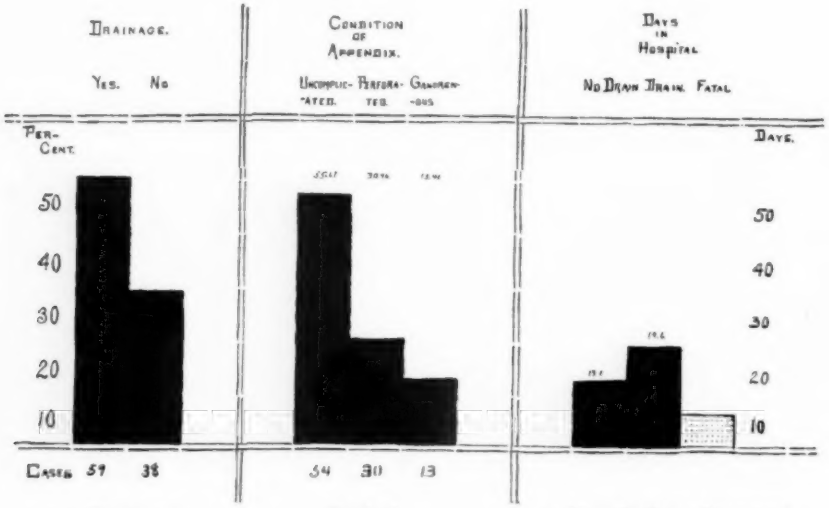
Eight of the 9 fatal cases had a blood count made just prior to operation, as one of the deaths occurred following an early operation for simple catarrhal appendicitis from an intestinal obstruction on the ninth day and happened to be the one case on which no count was made. This one will be put aside for statistical purposes, leaving 8 cases to be considered.

Only 3 had a white blood cell count of below 10,000 and one of these was 9800. Thus of the whole number, 14 cases had total

white cell counts of from 5000 to 1000, with a correspondingly low polymorphonuclear count; of these only 4 cases were fatal.

Group 2. Late cases: over twenty-four hours.

Group 3. Late cases: forty-eight hours,



A. Drainage. B. Pathology. C. Hospital confinement.

Fig. 4.

As regards the differential count: twelve cases out of the 97 had under 70 per cent poly morphonuclears of the fatal cases, 2 out of 8 had under 70 per cent polymorphonuclears. Ten cases with this count recovered.

This seems to agree with Hillweg's statement that "leucocytosis is a mere symptom, inconstant and unreliable."

Time. Now as regards the length of time from onset of pain until operation: In the 97 cases, this averaged 85.4 hours. The fatal cases averaged 198 hours. The average time of the cases that recovered was 79.4 hours. Thus the time element seems to be the most important one factor we have to deal with in considering the mortality of appendicitis.

After the first hours in an appendiceal infection, the pathological process does not necessarily progress at an even time rate and any classification as regards this time must be necessarily arbitrary.

For convenience, I have made a classification which, though crude, is fairly satisfactory for hospital ward use:

Group 1. Early cases: twenty-four hours.

complicated by local peritonitis.

Group 4. Late cases: forty-eight hours, complicated by general peritonitis.

According to this classification, we find the following:

Group 1. Early cases (twenty-four hours): 29 cases; 0 deaths.

Group 2. Late cases (over twenty-four hours): 26 cases; 1 death. (On the seventh day due to postoperative intestinal obstruction.)

Group 3. Late cases (over twenty-four hours, complicated by local peritonitis and abscess): 25 cases; 0 deaths.

Group 4. Late cases (over twenty-four hours, complicated by general peritonitis): 17 cases; 8 deaths (42.3 per cent).

In Groups 2 and 3 the condition of the appendix was definitely stated in 43 cases by the surgeon: Appendix perforated in 30 cases; appendix gangrenous in 13 cases.

Drainage. All of the late cases with local and general peritonitis were subject to drainage of some kind and degree; in all. In addition in 17 of the 26 late cases in Group 2, drainage was used. Thus all the deaths occurred in late cases with

general peritonitis in which drainage was used except the one in the case of recurrent postoperative intestinal obstruction.

5 cases of 10 years or under (average 7 years).

4 cases 40 to 50 years.

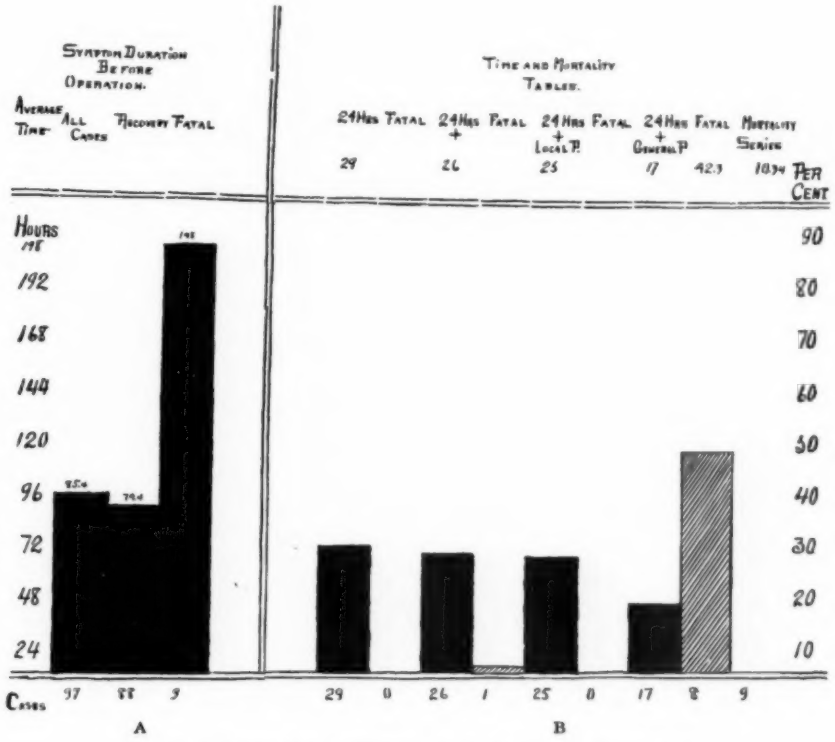


Fig. 5. A. Duration of symptoms. B. Time and mortality.

A variety of drainage means were used, the one most commonly employed being soft rubber tubes, often combined with Penrose rubber wick drainage. Penrose drainage alone was used by a small number of surgeons. The occasional operator always used rubber tubing.

Hospitalization. The average stay in the hospital of the drainage cases was 19.6 days; of the undrained cases, 13.1 days. One septic case developed pneumonia with complications which ran a long course. This case was hospitalized 40 days, which brought the average hospitalization up to 13.3 days. The hospital stay was shortest in the fatal cases, averaging 7 days.

MORTALITY

The total mortality of the series is 10.34 per cent. The total recoveries of the series is 89.66 per cent. The fatal cases may be tabulated:

6 patients were males.

3 patients were females.

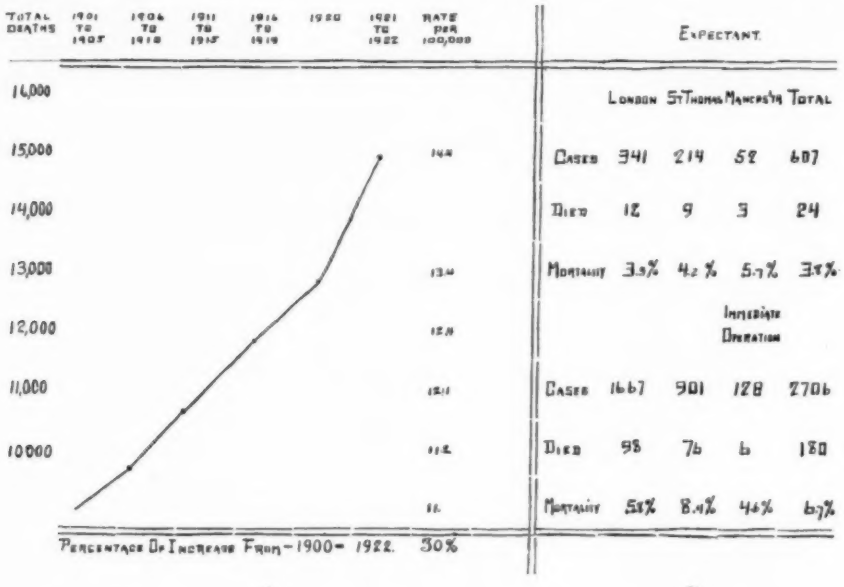
Average age of males was 22 years.

Average age of females was 15 years.

In an article by A. M. Willis[4] he states that from 1901 to 1922 the mortality of appendicitis increased almost 31 per cent. His chart illustrating this increase was compiled from the Table of Vital Statistics issued by the U. S. Government; it does not tell what percentage of cases were operated or unoperated on.

What would be the mortality if appendicitis were treated expectantly? This is of course a very difficult question. Fitz of Boston, whose memorable paper on this subject appeared in 1887, gives statistics showing a mortality of 25 per cent. During the 90's at every Medical Meeting, the discussion waged fiercely as to whether to consider appendicitis as the case for the surgeon or the medical man. Only a very few

of the surgeons dared claim that it was a surgical disease; most were content to wait until a tumor formed, then incise and drain. rest and a suitable diet, in which the mortality was only 16 per cent. These varying forms of treatment may explain



A B

FIG. 6. A. Willis chart of appendicitis mortality. B. English mortality tables.

That the frequency of appendicitis must have been approximately the same in those years is evidenced by tables published in 1893, showing 100 cases of peritonitis due to ruptured appendix out of 1800 post-mortem cases.

Robert Morris, of the "Morris sign" and the "Morris appendix," stated in 1899 that following medical treatment the death rate was at least 25 per cent and that under proper surgical treatment in the beginning of an attack the mortality should not be over 1 per cent. Maurice Richardson, who was one of the most skillful and beloved of Boston surgeons, this same year, however, reported 19 consecutive deaths following operation for appendicitis and questioned whether the surgical rate was not greater than the medical. Ewald in 1891 claimed 90 per cent would recover under medical care.

As a matter of fact, at and before this time there were two views regarding the treatment of appendicitis medically:

(1) By purgatives and injections, which gave in the reported series a mortality of 40 per cent; (2) under tincture of opium, the wide discrepancies in the claims of the mortality tables.

When I was an interne in 1895 there were still a number of practitioners who treated their appendicitis cases medically unless forced to call in a surgeon because of a walled-off abscess. One of the three surgeons then on the staff, a man of German birth and education, stated repeatedly: "Appendicitis is not so difficult. Opium every day . . . in two weeks, if it is ripe, a cut with a scalpel and . . . it is all over." In many cases his patients survived at least the current attack. The mortality in this hospital must, as I remember, have been about 15 to 18 per cent.

J. B. Murphy claimed on March 2, 1889 he operated upon the first case of appendicitis according to the modern procedure: i.e., deliberately opening the peritoneal cavity in the absence of phlegmon or pronounced induration of the abdominal wall. Murphy in 100 cases from 1889 to 1893 reported his mortality as 11 per cent.

Every death from appendicitis represents an error on the part of some one

connected with the case: the physician, the surgeon, the hospital, the patient or his friends.

In this series the mortality is due to the length of time elapsing from the inception of the symptoms until operation. A physician today should be able to make a diagnosis of appendicitis or at least a surgical condition within twenty-four hours and advise hospital care and operation.

The death rate today is higher than it was two decades ago, although we have learned much in that time. Twenty years ago appendicitis was a live topic. A majority of the profession was converted to the fact that no time should be wasted in having surgical care. The result was a 2 per cent death rate. Today with so many avenues opening up to claim the medical man's attention, I do not think he is so keen and decided about his views as he was then.

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EPISIOTOMY
AS A MEANS OF PRESERVING
THE PELVIC FLOOR DURING LABOR, WITH A SIMPLE METHOD OF SUTURE*

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ANATOMY OF THE PELVIC FLOOR

THE pelvic floor or pelvic diaphragm is a muscle-fibrous partition which closes in the abdominal cavity inferiorly. It closes the space known as the pelvic outlet or inferior strait, which is an oval space bounded anteriorly by the symphysis pubis, posteriorly by the tip of the sacrum and coccyx, and laterally on either side by the rami of the pubes and ischia and the great sacrosciatic ligaments. Just as the abdominal cavity is separated from the thorax by a muscle-fibrous diaphragm, which is pierced by apertures for esophagus, aorta, and vena cava, so is the pelvic outlet closed by a similar diaphragm which is also pierced by three apertures for the passage of the urethra, vagina and rectum. To these three structures, as well as to the bladder, uterus and adnexa above them, the pelvic diaphragm forms the main support and by its integrity prevents bulging, herniation or prolapse of the pelvic and abdominal viscera.

During parturition, however, as the child passes through the vagina, the structures of the pelvic floor are subjected to a great amount of stretching and stress, which in the majority of cases it seems unable to withstand, and which results in more or less separation of its attachments, tearing of its fascia or rupture of its muscular fibers. As soon as the structures are torn the ends separate, forming a gap similar to that which takes place in the severance of a tendon or muscle elsewhere. This results in relaxation or sagging of the pelvic diaphragm with consequent displacement, prolapse or herniation of the organs to which it gives support and relative amount of disability to the patient, depending on the temperament of the patient, the extent of the damage done and the skill with which it has been repaired.

In order to better indicate the structures involved and the changes which occur as a result of injury during delivery, the following facts concerning the anatomy of the pelvic floor or diaphragm should be kept in mind. This properly includes all the structures which close in the pelvic outlet from the peritoneum above, to the skin below. It consists of two distinct layers of muscles and their fascial coverings.

The inferior layer, derived from the primitive cloaca, includes the deep and superficial transverse perineal muscles, the bulbocavernous, the ischiocavernosus, and the constrictor urethrae. These muscles are for the most part sphincters in action and have little or no supporting function.

The upper layer forms a broad sheet of muscle and fascia consisting of the levatores and the ischiococcygeus muscles on either side, the latter forming the posterior portion of the pelvic diaphragm while the levator ani forms the remaining lateral and anterior portions, except for a short space on the posterior aspect of the symphysis pubis, which is closed in by the triangular ligament. The levator ani is composed of two separate muscles. The external muscle, the iliococcygeus, arises from the rami of the pubis and the pelvic fascia of the white line as far back as the margin of the great sacrosciatic notch. Its fibers are inserted into the sacrum, coccyx, and anococcygeal raphe. The fibers pass from the two sides and decussate

* Presented as a Thesis to the Fellows of American Association of Obstetricians, Gynaecologists, and Abdominal Surgeons, Memphis, Tenn., Sept., 1929.

with one another in the space between the anus and coccyx. The internal portion of the levator, the pubococcygeus, arises

Fig. 1. Showing deep dissection of pelvic diaphragm with horseshoe-shaped margin of levator ani muscle with decussation of anterior fibers in perineal body (fibers of Luschka). Cut edge of rectovesical fascia as it rounds anterior free border of muscle to fuse with anal fascia (which has been removed), is seen. (Redrawn after Kelly.)

from the fascia on the back of the symphysis pubis, the pubic rami and from the obturator fascia. The outer and upper third is attached to the sacrum and coccyx; the middle portion, the puborectalis, decussates with its fellow of the opposite side to form a sphincter which slings the rectum to the symphysis pubis and is that portion of the levator most effective in lifting the rectum; the muscle fibers of the innermost third pass behind the vagina without insertion into the vaginal wall to join the longitudinal fibers of the rectum and the external sphincter ani, partly by direct union and partly by decussation with fibers from the opposite side in the space separating the anterior surface of the rectum from the posterior surface of the vagina—the so-called median raphe of the perineum, the muscle-tendinous spot or the central body of the perineum, etc. This internal portion of the levator ani forms, with its fellow of the opposite side, a sling for or constrictor of the vagina posteriorly and is the portion subjected to the greatest amount of stretching and strain during labor. (Fig.

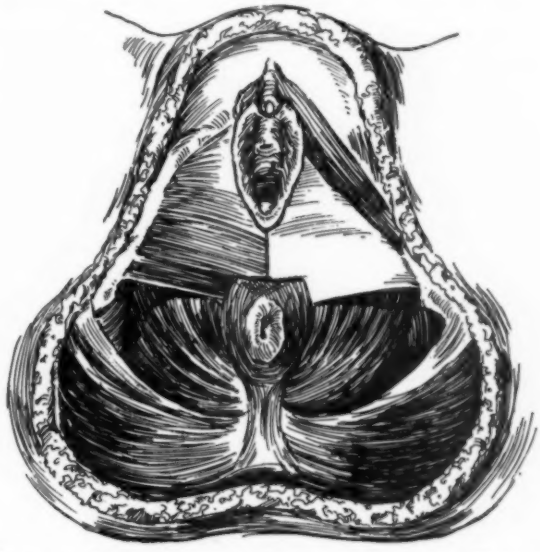

Fig. 2. Urogenital trigone or triangular ligament closing in pelvic outlet anteriorly and pierced by vagina and urethra. (Redrawn after Kelly.)

1.) Injury to the fascia and muscle in this location results in many cases from the overstretching incident to even natural labor and may result in separation of its attachments to the median raphe and rectum, with diastasis, sagging of the anterior rectal wall, and the later development of rectocele.

The levator muscle is covered by fascia, the upper surface by the rectovesical fascia, fascia supradiaphragmatica, or the visceral layer of pelvic fascia which completely closes in the pelvic canal except for the openings of the urethra, vagina and rectum. The lower surface is covered by the anal fascia, or fascia infradiaphragmatica, forming the inner wall of the ischiorectal fossa and blending with the perineal and anococcygeal raphes. At the free border of the levator ani muscle, as it encircles the vagina laterally and posteriorly, the upper rectovesical layer fuses with the lower anal layer. This forms a fascial sling between the rectum and vagina and as has already been stated, this is the portion subjected to the greatest stress and strain during labor and when

injured is directly concerned with the formation of rectocele.

Closing in the open space between the pubic rami and the inner border of the levator bundles is the urogenital trigone or triangular ligament. It is composed of two strong fascial layers attached in front and at the sides to the pubis and ischiopubic rami as far back as the ischial tuberosities. Posteriorly and laterally it lies below and overlaps the levator muscle and its fascia. Its upper layer blends with the lower layer of the levator fascia. Between the two layers of the triangular ligament, lie the deep transverse perineal and the constrictor urethrae muscles. The urogenital trigone is pierced by the urethra and vagina, to both of which, as well as to the bladder and uterus above them, it gives considerable support, supplementing that of the rectovesical fascia above. Overstretching and laceration of the triangular ligament promote relaxation of the anterior segment of the pelvic floor with the formation of cystocele, urethrocele and prolapse of the anterior vaginal wall. (Fig. 2.)

INJURIES TO THE PELVIC FLOOR FROM PARTURITION

For practical purposes therefore as may be seen from the foregoing description, the structures of the pelvic floor may be arbitrarily divided into two segments; an anterior segment closing in the triangular area between the pubic rami composed from above downward of the anterior half of the rectovesical fascia and below the double layer of the triangular ligament; and a posterior segment composed chiefly of the levator ani muscle and the fascia covering its upper and lower surfaces. As the shape and dimensions of the pelvic outlet are subject to wide variations so are these two segments of the pelvic floor subjected to widely different amounts of strain and damage. While in a general way, this may be considered to depend on several factors, such as the size of the child, the age of the gravida, the elasticity of the soft parts, the amount of scar tissue from previous repairs and the presence or absence of edema of the parts, the two most important factors to be considered are firstly the size of the baby's head and secondly the width of the pubic arch and the transverse diameter of the pelvic outlet. On the one hand, when the pubic arch is wide and the distance between the tubera ischi greater than 8 cm. the presenting parts fit up well between the pubic rami and closely under the symphysis, causing the anterior segment of the pelvic floor to be subjected to the greater amount of stretching and possible injury, with the subsequent development of cystocele, urethrocele, and prolapse of the anterior vaginal wall and bladder; whereas, on the other hand, when the pubic arch is narrow and the transverse diameter of the outlet contracted to less than 8 cm. as is found in the so-called funnel type of pelvis, the presenting part is displaced posteriorly and delivery then takes place more at the expense of the posterior segment of the pelvic floor. In this instance one would expect a greater incidence of injury to the sling of the pubococcygeus bundle of the levator and muscle and its fascia with the subsequent formation of rectocele and furthermore rupture posteriorly of the perineal body, vaginal sulci and occasionally the external sphincter ani and rectal wall.

Tissue reaction to stretching and strain depends to a great extent on the length of time allowed for the stretching to occur, or in other words on the rapidity or slowness with which the force is applied. That the tissues of the modern woman do not well withstand the tension and stretching incident to the average normal labor and that injuries to the pelvic soft parts occur in the great majority of so-called normal labors is a well-known fact. How much greater must they be, however, when the amount of the force is greatly increased in intensity and applied and exerted with an unnatural suddenness

which does not allow the tissues time to stretch as obtains in too rapid delivery with strong bearing down effort on the part of the patient without the relaxation of anesthesia, too rapid manual dilatation or breech extraction, the misuse of pituitrin to hasten delivery and the unnecessary and unskilled use of forceps, especially when they are used to rotate the head from a posterior or transverse to an anterior position. This may result in stripping the levator from its attachments to the pelvic wall or in deep sulcus tears, occasionally laying open the ischiorectal fossa, the fatty tissue of which can be seen in the rent on the side subjected to the greatest amount of strain by rotation. Williams[1] states that injuries to the perineum are of very frequent occurrence, and cannot always be avoided even under the most skillful treatment. Statements as to their frequency vary considerably from those of the physician who has not had a single tear in a 1000 cases, to those who find more or less damage in more than 75 per cent of their primiparous labors. In the former case one would class the attendant as a rather poor observer, because as estimated by Williams,[1] lacerations occur in about 40 per cent of primiparous labors and in about 10 per cent of multiparous labors. These figures undoubtedly are given for lacerations characterized by open wounds where the vaginal mucous membrane or skin is torn along with the underlying structures. Such a laceration is easily recognized and in the majority of cases can be satisfactorily sutured. Much less fortunate are those patients in whom the vaginal integument and skin are not torn but in whom there have been submucous lacerations of the deeper structures of the pelvic floor or separation of the attachments of the levator to the median raphe and rectum. It is impossible to recognize such an injury at the time of labor and these patients are discharged with relaxed supports to the pelvic viscera, greatly enlarged vagina with loose sagging walls and a relaxed, gaping introitus. Moreover laceration of the anterior segment of the pelvic floor including rectovesical fascia and the triangular ligament with the formation of cystocele is a more or less irreparable injury in a young woman and if repaired is likely to recur during subsequent delivery.

SCOPE OF EPISIOTOMY IN PREVENTION OF INJURY

To discharge a patient at the end of the puerperium as well as she was before she became pregnant is the supreme test of the competent obstetric practice and the only one by which our work should be judged. This well-being or fitness applies as well to the integrity of the perineum and pelvic floor, the size of the vagina and the proper supports of the pelvic organs as to the general constitutional health of the mother and child. That this is not accomplished and that the methods of preventing lacerations, and guarding the perineum described in textbooks are inadequate is mutely but forcibly testified to by the large number of women who flock to the gynecological clinics of the country for the repair of injuries incurred at childbirth and who state as the most significant fact in the history of their disability, that they have never been well since the birth of one of their children.

Kelly[2] states that "any considerable injury to the perineum means a laceration of the perineal fascias deep and superficial, the urogenital diaphragm, the levator fascia, and the anterior fibers of the pubococcygeus bundle of the levator ani muscles with consequent retraction of the muscle and separation of the torn ends. In the laceration of long standing there is always atrophy of disease." Pomeroy[3] states:

> Every primipara incurs a permanent modification of the pelvic floor in the course of a full-term delivery. In a high percentage of cases this is represented by an open lacerated wound and in nearly all of the others concealed damage to the levator ani muscles and fascia

is acknowledged to occur and to be the factor paramount in the various degrees of subsequent prolapsus uteri, cystocele and rectocele. We, as gynecologists, are devoting years of thought and ingenious labor to planning and executing operations for the repairs of injuries due to childbirth; but so far obstetricians have not faced and accepted a reasonable responsibility for the discovery of a plan to prevent, by sound surgical procedures serious birth divulsion damage to the structures of the pelvic floor. The general attitude toward saving the perineum in the second stage of labor as advocated by textbooks of obstetrics is that little can be done except encouraging gradual dilatation by the avoidance of precipitate expulsion or too rapid extraction in unfavorable positions.

Pomeroy[4] concurs with others in the idea that: "Rending the birth canal to enlarge its caliber is strictly unsurgical." In no other phase of the practice of medicine or surgery are tissues allowed to be bruised, overstretched and rent apart as they are in childbirth, when the same end can be more easily and directly accomplished and more in keeping with surgical principles by cutting the tissues before tearing or divulsion occurs. This may be well accomplished by the operations of episiotomy or perineotomy which is an incision of the posterior vaginal wall and the structures of the perineum and pelvic floor and the restoration of the parts to an integrity similar to that of the nulliparous state after suturing, which is easily performed in a clean-cut, superficial wound. That it accomplishes this end in the majority of cases may be deduced from the description of the anatomy and relations of the structures involved. If the incision is made deeply enough and at the proper time, when the vaginal orifice is dilated to a diameter of about 4 cm. and when the structures of the pelvic floor and perineum, including the sphincter ani, have been well stretched and not overstretched, the incision will sever the following structures, namely, the vaginal mucosa, the united free border of the rectovesical and anal fascias, the innermost and shortest portion of the pubococcygeus bundle of the levator muscle, especially where it decussates in the space between the rectum and vagina, the posterior portion of the triangular ligament, the superficial perineal muscles, the superficial fascia or fascia of Colles, and the skin. The incision may be made in the midline as in median episiotomy or laterally or bilaterally as in oblique, lateral or bilateral episiotomy. The median incision is to be preferred in the majority of cases because it passes through the stronger instead of the weaker structures, relieves tension symmetrically, and provides more ideal conditions for symmetrical reconstruction by suture. The only assailable point in the claim for superiority of the median incision is the risk of injury to the sphincter ani and of extension backward through the anal and rectal wall. This risk can be greatly minimized by having the anal sphincter thoroughly stretched and paralyzed before making the incision, or when in doubt, as in cases of funnel pelvis or when the child is disproportionately large, by using oblique incision. A relaxed sphincter cut in the midline is usually quite accessible for repair, easily sutured and heals promptly. Episiotomy, by cutting these structures either in the midline by median episiotomy or laterally by cutting through the belly of the muscle on one or both sides as in oblique, lateral or bilateral episiotomy, greatly diminishes the tension on the deeper structures of both the anterior and posterior segments of the pelvic floor, prevents submucous and subcutaneous lacerations and when properly sutured, restores the parts in the majority of cases to their original state.

REVIEW OF CASES

In a review of the last 500 consecutive cases of median episiotomy performed by the writer, primary healing of the wound and a satisfactory end-result were obtained in all cases. In the majority of them the integrity of the pelvic floor seemed

intact while in a small number there were slight or moderate degrees of relaxation of either the anterior or posterior segments.

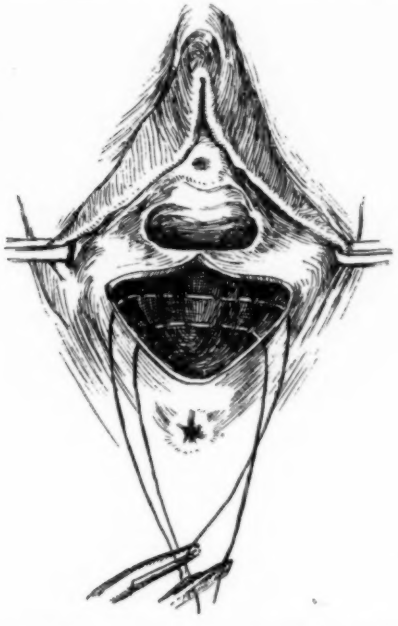

Fig. 3. Median episiotomy wound with sutures buried deeply into retracted severed attachments of levator ani muscle, rectovesical and anal fascias and urogenital trigone.

Six cases showed superficial infections or stitch abscess which did not in any way mar the end-result. Febrile puerperia occurred in 65 cases, 41 of which were attributable to incidental or intercurrent causes as bronchitis, mastitis pyelitis, etc. This gives a total morbidity from all causes of 13 per cent and a corrected morbidity of 4.8 per cent. Considering infection of the wound alone as a cause of morbidity, there were 6 infected wounds in all or 1.2 per cent.

There were 9 cases of bilateral extension up the vaginal sulci and 9 cases of unilateral extension. The majority of these occurred in forceps deliveries either before or after the incision of the perineum. The anterior fibers of external sphincter ani were cut or injured in 15 cases. Extension through the external sphincter occurred in 16 cases and extension through the sphincter and involving the anal margin or rectal wall in 8 cases or 1.6 per cent. With more attention to preliminary dilatation of the external sphincter and stretching of the rectal wall before making the incision and also by diverting the incision to one or the

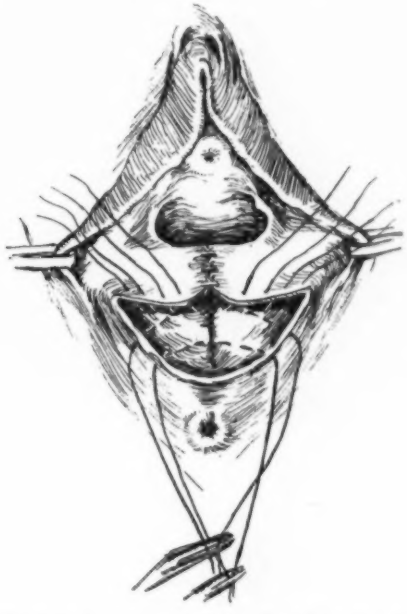

Fig. 4. Vaginal mucosa and underlying fascia closed with interrupted catgut sutures.

other side when extension backward seems imminent, the incidence of sphincter injuries and complete tears can be reduced.

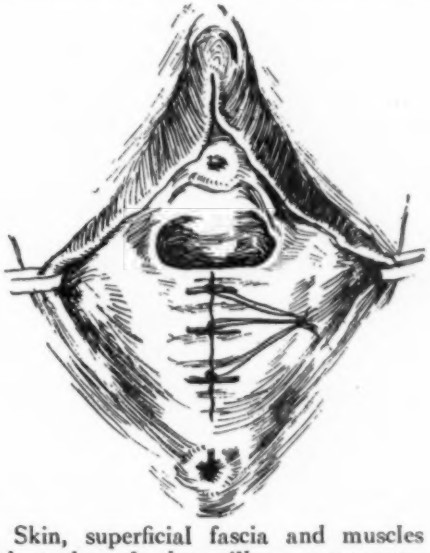

Fig. 5. Skin, superficial fascia and muscles closed with moderately deep silkworm gut sutures.

Where the pubic arch is narrow and the child of considerable size, or when there is edema of the vulva and perineum, it is safer to make an oblique incision one

side of the sphincter ani. Four of the cases with third degree tears had febrile puerperia and one a non-hemolytical streptococcus septicemia, but all healed primarily and in the end made good recoveries. There were 6 cases of phlebitis, 4 of which had pulmonary infarction. There were no deaths in the series.

Old lacerations were repaired through the episiotomy wound in 67 cases, second episiotomy on the same patient at a subsequent delivery in 75 cases, 3 episiotomies on the same patient in 11 cases and 4 episiotomies with same patient in 4 cases. Four patients complained of tenderness in the region of the perineum for several months after delivery but otherwise the results seemed almost uniformly good.

A SIMPLE TECHNIC FOR REPAIR

The wound is usually sutured immediately after delivery of the child while the patient is still anesthetized and before separation of the placenta has taken place. No ill effects from delivery of the placenta after the sutures have been placed and tied have been noted.

The introitus is retracted on either side by a Gelpi retractor or tenacula or sutures held by an assistant. Two or three sutures of twenty-day catgut are then placed deeply in the tissues of the deeper plane which should include the severed attachments of the levator bundles with its fascia and urogenital trigone. The sutures are inserted deeply to bring back the structures, which have retracted as a result of their own elasticity, and are introduced at right angles to the course of the muscle fibers and fascial planes. (Fig. 3.) The sutures are clamped and left long to facilitate closure of the vaginal mucosa. This is done with interrupted or submucous catgut sutures including the fascial plane beneath the mucosa. (Fig. 4) The deep buried sutures are then tied and the skin of the superficial muscles and fascia are closed with interrupted silkworm gut. (Fig. 5.)

CONCLUSIONS

1. More or less damage to the pelvic floor is caused by delivery in the majority of women.

2. Episiotomy affords an efficient means of lessening the extent and ill effects of this damage.

3. In the proper environment, episiotomy does not seem to increase the morbidity or risk of delivery.

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4. Pomeroy, R. H. *Am. J. Obst.* 78: 212, 1918

AN ADJUSTABLE EXTENSION FOR FRACTURES OF THE UPPER THIRD OF THE FEMUR*

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AS may be noted in the accompanying photographs the adjustable extension for fractures of the upper third of the femur consists essentially of two parts, a stationary plate which is readily attached to any bed simply by the quick turning of two clamps, and a movable arm which describes a circle on which arm a pulley travels throughout its length.

FIG. 1. Fractured neck of femur. Rear view of extension showing how elevation and abduction are maintained. Dotted line indicates circle of tension or circumference within which tension may be applied.

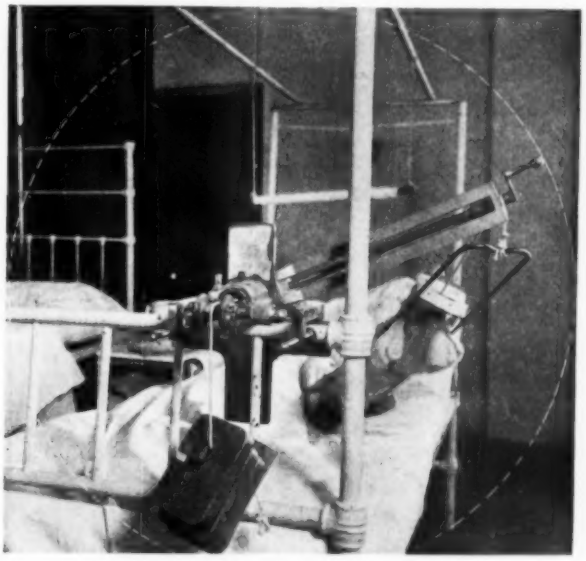

FIG. 2. Rear view of extension.

The extension is adjustable in all directions so that the part under tension may be placed in any position at any time without altering the amount or the line of tension desired. The fractured extremity may gradually be placed in the position of elevation and extreme abduction. Daily dressings, more particularly of the under surfaces, are readily accomplished. Decubituss ores are easy to approach. The patient himself has a fair amount of freedom facilitating bedside care considerably. Passive motion may be instituted early without danger of the fractured fragments over-riding.

The finest of adjustments obtainable with this device permits the most careful placing from time to time of fractured parts, with or without roentgenological aid. Degrees of angulation, inches and centimeters are noted on the instrument. Although this device has been found particularly adaptable in fractures of the upper third of the femur, it is also ideal for the treatment of other fractures, dislocations, sciatica or any other conditions requiring the application of extension.

Sixty cases of fractures of the upper third of the femur have been treated successfully at the Albany Hospital[1] with this device.

[1] Service of Dr. Arthur Elting.

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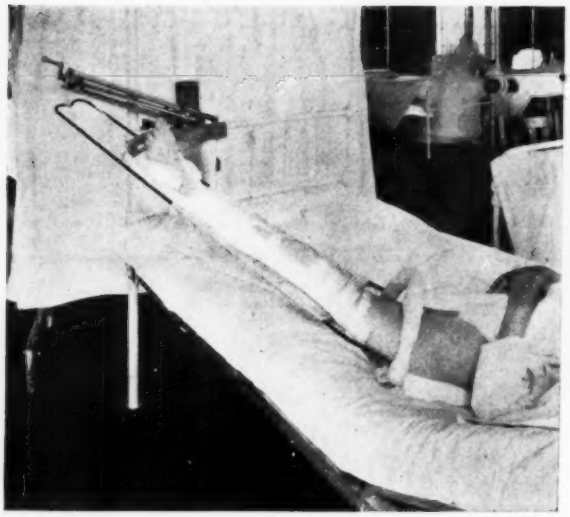

FIG. 3. Front view of extension. Thomas splint in use.

FIG. 5. Extension applied to regular bed at home.

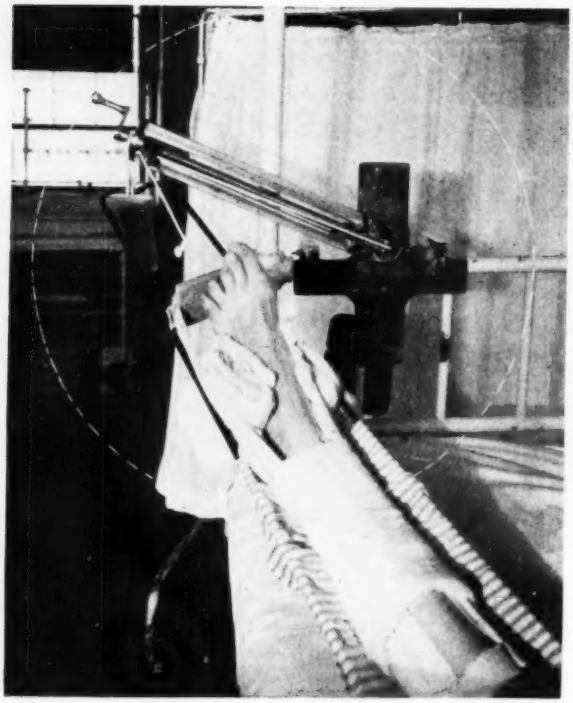

FIG. 4. Front view of extension.

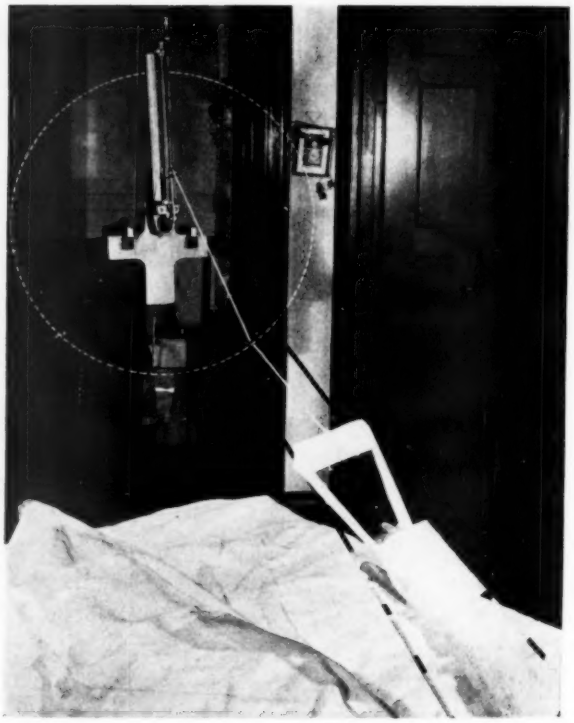

FIG. 6. How extension may be applied to fractured upper extremity.

FIG. 7. Comminuted intertrochanteric fracture of right femur in patient aged fifty-five.

FIG. 8. Same patient nine weeks later, able to walk unaided.

CHRONIC INFLAMMATORY VESICAL NECK OBSTRUCTIONS

A CLINICAL AND ANATOMICAL STUDY*

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A PERUSAL of the literature will show that there is considerable confusion in our conception of median bar, due not only to the fact that the term median bar includes obstructive elevations of fibrous and glandular types, which of themselves are separate entities, but also due to visualization of pathology through the use of different types of diagnostic cystoscopic instruments.

Diverse types of pathology are still grouped under the original descriptive term suggested by C. J. Guthrie in his lecture on median bar, delivered before the Royal College of Surgeons in London, in 1830, nearly one hundred years ago. In 1850, Mercier, in his report, described the same condition although his name has often been erroneously associated with the elevated interureteric ridge. From this date to the beginning of the present century, no really important advances can be said to have been made in the clinical or pathological conception of this subject. Since then, the work of Zuckerkandl, Alberran, Chetwood and others, followed by Lowsley's careful embryological and anatomical studies of the prostate and its contiguous structures, the thorough work of Randall on the prostate, subcervical glands and bladder neck, and the anatomical studies of the internal sphincter and vesical neck by Young and his associates, have aided materially in clarifying our understanding of the fibrotic bar and sclerotic vesical neck obstruction.

The fibrous median bar is best described as a cicatricial obstruction caused by fibrotic elevation of the posterior vesical lip with or without coarctation and constriction of the entire circumference of the vesico-urethral orifice. The relation of fibrous median bar formation to deep seated cicatricial changes in the vesical neck and sphincter, urethral changes as well as those taking place in the prostate and seminal vesicles, has not been clearly correlated. If an infection be superimposed upon one or more of these uncorrelated structures, we may logically assume that other structures in relation to those infected will likewise become involved. Variation in anatomical structure has a direct influence on the pathogenesis. These are important points to bear in mind, not only on the diagnosis and treatment, but on the morbidity of this condition.

Because of the correlation of objective instrumental and operative observations with microscopic studies of the excised surgical specimens and routine necropsy specimens, our understanding of the gross and microscopic pictures is clear and definite. However, the problem of pathogenesis and etiology is still a moot question. The pathogenesis of a condition such as contracture must be obscure as long as the different stages in the production of terminal pathology cannot be demonstrated. The initial lesion cannot be known and appreciated without a definite conception of the pathogenesis and therefore the etiology is difficult to deduce.

In an effort to arrive at a better understanding of the underlying morbid processes of this condition, we have carried on, for the past year, a study of its pathogenesis and etiology. In this work, microscopic studies were made of 10 surgical punch specimens removed from clinically and cystoscopically proven cases of median bar or vesical neck contractures, with the McCarthy visualized punch. Parallel with this, gross and microscopic observations were made of the bladder, vesical neck and posterior urethra, and adnexa of 50 fresh cadavers selected from several hundred necropsies, while at the same time

* Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, December 18, 1929.

cystoscopic observations were made of clinical patients presenting early and late symptoms and physical signs of vesical neck obstruction. On fresh necropsy material, visual and palpatory evidence of elevations, nodules, and contractures of the vesical neck was classified according to Randall's types of contracture or fibrous bar. From specimens showing this type of deformity transverse and sagittal serial sections were made of the posterior urethra, vesical neck and trigone, including the contiguous prostate and seminal vesicles. This enabled us to perceive the underlying pathology in the posterior urethra, prostate and seminal vesicles in necropsy material which was compared with the microscopic picture of surgical specimens removed by means of the punch in proven clinical cases. Where cases of acute, subacute and chronic infection of upper and lower urinary tracts were encountered, similar serial sections of the posterior urethra, vesical neck and trigone were made. By noting the changes in the vesical neck structures in all stages of inflammation of the posterior urethra, prostate and seminal vesicles, accurate deduction could be made of the underlying etiology and pathogenesis of bladder neck fibrosis.

In addition to the well developed prostatic glands, which empty on the floor of the posterior urethra, a homologous group of superficial glands exist in the posterior urethra, vesical neck, and trigone (Fig. 1). In many cases, they are rudimentary or absent. In other instances, they extend for varying distances into the mucosa and submucosa, and occasionally the muscularis. In the posterior urethra these glands predominate on the sides and roof. On the vesical neck and trigone, they are almost always limited to the central portion of the posterior vesical lip. Although they are given a variety of names, we shall call them submucosal glands of the posterior urethra, vesical neck and trigone. We shall later demonstrate the importance of these structures in the development of submucosal fibrosis.

Pseudo-adenomatous hypertrophy of the submucosal glands of the vesical neck or of the glands of the posterior prostatic

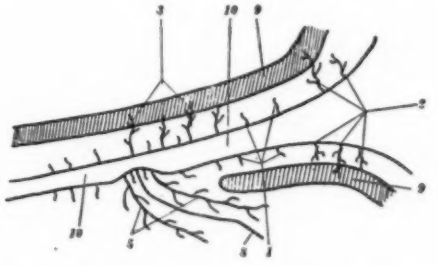

Fig. 1. Schematic representation of glandular structures of posterior urethra, vesical neck and trigone. (From Jacoby.) 1, Short mucosal glands of posterior urethra and vesical neck. 2 and 3, Submucosal glands of posterior urethra, vesical neck and trigone, reaching superficial layer of muscularis. 5, Prostatic glands arising in the premontane and postmontane urethra. 8, Ejaculatory duct. 9, Muscle of posterior urethra, vesical neck and trigone. 10, Urethral lumen.

commissure, may cause elevation of the posterior lip of the vesical neck. However, they are usually excluded when fibrosis of the vesical neck is considered, except in those rare instances where a combination of both factors exists. In the accepted conception of the pathology of vesical neck cicatrizations, inflammation is believed to account for all those cases classified as acquired. Fibrosis of the retrogressive type associated with seniel changes and arteriosclerosis, is not considered as important. Primary congenital muscle hypertrophy of the sphincter with secondary fibrosis due to congestion and infection accounts for some of the cases reported.

The excised surgical specimens in our series on the basis of the history, clinical and microscopic findings, conformed with the accepted views. One specimen, clinically and microscopically, suggested congenital hypertrophy of the sphincter with superimposed superficial, submucosal, active, chronic inflammation (Fig. 2). All the other specimens showed microscopic changes evidently due to acquired inflammatory causes, and clinically consisted of (1) cases of typical findings of contracture or bar; (2) cases of atonic bladder without a neurogenous cause; (3) one case of atonic

bladder in which a destructive spondylitis, aroused suspicion of a neurogenous element. The microscopic picture of this urethral inflammation, as well as typical vesical neck contractures and bars, it was possible to find changes in the superficial

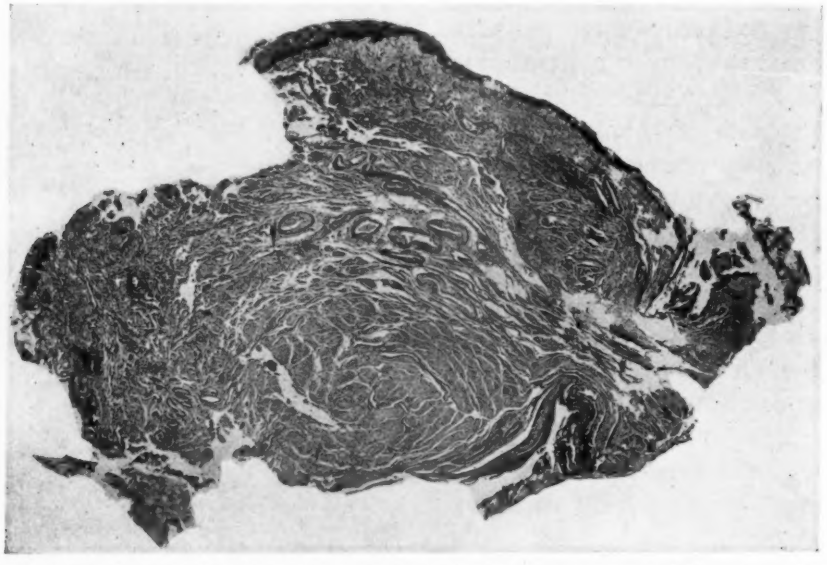

Fig. 2. Low-power photomicrograph of punch specimen classified as congenital hypertrophy of sphincter with active chronic inflammation in submucosa. Large vessels in submucosa indicate long-standing congestion.

group of cases left no doubt as to the inflammatory factor (Fig. 3). In addition to fibrosis in the submucosa and disorganization of the muscularis by scar tissue, accumulation of round cells was found in the submucosa and muscularis, frequently perivascular and periacinar. Glands seemed to play an important rôle in the inflammatory process in some cases, because of the depth of their penetration into the muscularis and their evident cystic dilatation and intra-acinar and periacinar cellular exudation. All stages of inflammation were noted, from an active and recent process, to a quiescent stage of scar in which glandular elements and muscle tissue were similarly embarrassed by the fibrous overgrowth.

Based upon clinical and operative studies of cases of median bar and sclerosis of the vesical neck, and very infrequent and inadequate post-mortem examinations, the associated pathology in the contiguous centripetal structures must be a matter of conjecture. In the course of routine microscopic serial studies of the vesical neck and contiguous structures in necropsy specimens showing all stages of posterior structures which corresponded to the microsopic findings in the excised surgical specimens. Where this occurred, the contiguous structures of the posterior urethra, prostate and seminal vesicles were subjected to careful study. By noting the effect of acute and subacute inflammation of the posterior urethra and adnexa upon the vesical neck, the preceding stages in the development of contracture of the vesical neck could be followed. In addition, the deep seated changes in the prostate and seminal vesicles could be studied in those necropsy specimens which were manifestly the last stages of fibrosis.

By a study of our material a definite conception of the pathogenesis was formed. Acute inflammation, caused by infection or mechanical irritation, results in vesical neck changes which may be regarded as an acute inflammatory bar, because of the edematous appearance of the vesical neck and elevation of the floor. This is found associated with acute posterior urethritis, prostatitis, seminal vesiculitis, and less frequently with descending urinary infection. The submucosa of the posterior urethra is infiltrated with acute inflamma-

tory exudate which is also seen in the fibromuscular stroma of the prostate. The prostatic glands show hyperplasia with marked intra-acinar and periacinar exudation. The mucosal and submucosal glands of the posterior urethra, vesical neck and trigone are swollen, hyperplastic and filled with acute inflammatory cellular exudate, with corresponding acute exudation and infiltration in the superficial supporting tissues (Fig. 4).

Fig. 3. Low-power photomicrograph of punch specimen, typical of acquired inflammatory type. Marked fibrosis and round cell infiltration of submucosa. Muscularis shows marked perifascicular and intrafascicular fibrous and round cell invasion especially of superficial layers; chronically inflamed, cystic glands seen extending rather deeply into muscularis.

Due to continued infection, congestion or irritation of these structures, a stage of subacute or active chronic inflammation ensues. In this class of specimens, changes were noted which corresponded to some of the excised surgical tissues. In addition to typical chronic prostatitis and seminal vesiculitis, the superficial glandular structures of the vesical neck and trigone showed hyperplasia, cystic dilatation, periacinar and intra-acinar exudation with round and plasma cells. Frequently a rather thick layer of granulation tissue in different stages of organization was noted in the submucosa, and in the vesical neck and trigonal regions the superficial muscle layers showed similar changes. This highly vascular fibrous reaction is unquestionably the stage preceding that of actual cicatrization of the submucosa and muscularis of the sphincter and trigone (Fig. 5).

In the final stage, corresponding to that of the excised surgical specimens, a gradual transition is seen from active chronic inflammation of the superficial glands and their supporting tissues, to the picture of fibrosis with definite evidence of chronic

inflammation which has become quiescent. In many cases, a moderate number of cystic or hyperplastic submucosal glands

Fig. 4. Transverse section of low magnification through entire prostate and vesical neck of case of prostatic abscess. Higher magnification revealed concomitant acute inflammatory exudation in and around superficial submucosal glands.

may be found in the fibrous tissue replacing the sphincter muscle. In some cases, compressed glands were noted, and in others no glands were seen. In none of the cases were evidences of inflammation absent, and from the studies of the preceding stages, it can be said that the superficial gland structures of the vesical neck play an important rôle in determining the degree and depth of the inflammatory reaction in the submucosa and muscularis, even though this may not be evident in the late stages.

In some of these specimens of frank vesical fibrosis the prostate showed a corresponding advanced sclerosis with increase in the interacinar fibrous tissue and compression of the glandular elements. In others, only mild quiescent chronic inflammation was noted in the prostate and seminal vesicles (Fig. 6).

In most of the specimens illustrating the final stage of sclerosis, there was a definite localization of the fibrosis to the region of the posterior vesical lip. In others, the process also involved the sides and roof, but rarely to the extent that was noted on the posterior vesical lip. From our studies, it would appear that the cause for this localization is definitely due to anatomical structure, the submucosal glands of the vesical neck and trigone predominating on the posterior aspect of the vesico-urethral orifice. Productive changes in the stroma of chronically inflamed submucosal glands and subsequent cicatrization explains the fibrotic transverse elevation of the floor of the sphincter which causes obstruction (Fig. 7). The conception of this entity as a stricture of the vesical neck due to a deep seated sclerosis of the surrounding prostate is not satisfactory, since it cannot explain the transverse fibrotic elevation limited to the floor of the neck, and also fails to account for the splendid results which follow the removal of a relatively small piece of superficial tissue from the posterior vesical lip. When the coarctation is due to a small sclerotic prostate, minor surgical procedures are of no avail.

The diagnosis of vesical neck obstruction in the surgical cases was based on bimanual rectal palpation of the prostate and examination with the cysto-urethroscope, its forward vision giving an undistorted view of the posterior urethra, sphincter and bladder, and permitting an accurate estimation of the relative amount of elevation of bar formation and sphincteric change. In many instances it was supplemented by the use of the McCarthy panendoscope, not only to obtain the forward and lateral view, but as an added means of gauging the elasticity of the sides and roof of the internal sphincter. In every case, a complete urological examination was made which included detailed history, physical examination, and such roentgen and laboratory diagnostic measures as were necessary for a complete diagnosis.

While not always present in each of the foregoing cases, the presence of the obstruc-

tion at the floor of the vesical neck was often suspected when introducing the instrument, when it was found necessary to the verumontanum to a more rounded crest. Shortening of the retromontane urethra with an appearance of excavation

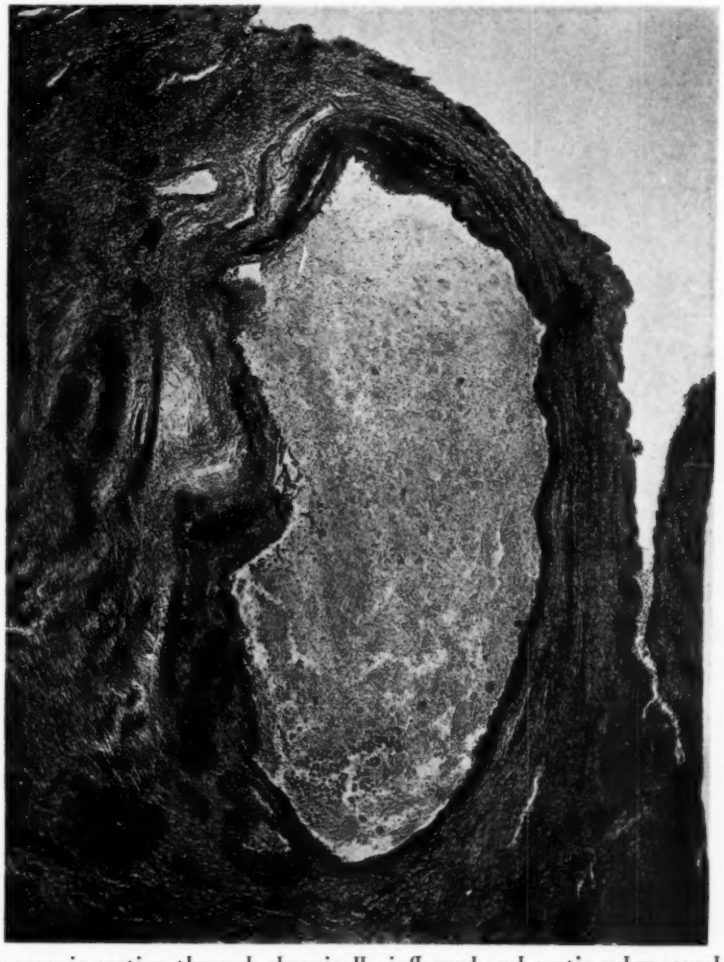

Fig. 5. Low-power microscopic section through chronically inflamed and cystic submucosal gland of vesical neck region. Active chronic inflammation also seen in supporting stroma. This pathological picture represents intermediate stage of chronic inflammation preceding that of acquired inflammatory vesical neck fibrosis.

to elevate the tip at the internal sphincter. Cystoscopically there was dilatation of the bladder in one instance, marked trabeculation in all, with sacculations and diverticula in many. The presence of bas fond was constant, as well as depression or elevation of the trigone. Elevation of the sphincter floor varied from a thin to a dense transverse fold, sufficient to obscure the interureteric ridge when viewed from the urethral aspect of the bar. The postmontanal portion of the posterior urethra was seen arising almost vertically to the uppermost portion of the bar, while in some instances, it made a vertical ascent from a depression immediately posterior on the urethral aspect of the sphincter was observed in several cases. The appearance of the mucosa, prostatic ducts and verumontanum showed no acute or subacute inflammatory change, but evident terminal fibrosis. Residual urine ranged in amounts from 4 to 44 oz.

Repeated cystourethroscopic examinations have impressed us with the fact that the vesical neck changes as seen through the cystourethroscope in these patients with fibrotic bar and sclerotic vesical neck might be considered as terminal pictures of many of the cases which are so frequently observed in practice, and who are being treated for chronic

posterior urethritis with associated chronic prostatitis, vesiculitis, and descending infections of the urinary tract. All types are

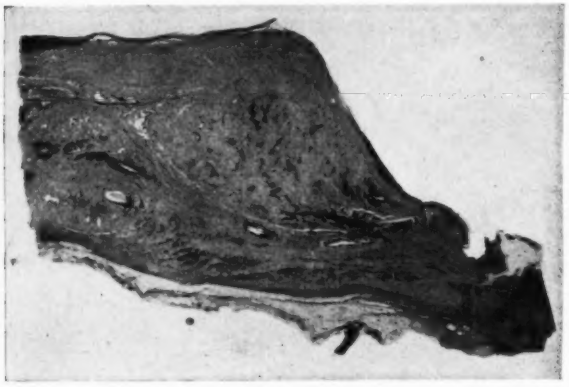

Fig. 6. Longitudinal section of floor of posterior urethra and vesical neck extending from verumontanum to interureteric ligament. Prostate shows marked increase in interacinar fibrous tissue with compression of glandular elements. Seminal vesicles show advanced sclerosis. A well defined group of submucosal glands seen extending into floor of sphincter which also shows marked fibrous invasion.

seen cystocopically in these prefibrotic tissue changes: the acute, subacute and early chronic stages. The sphincter floor does not show a dense transverse elevation of the posterior vesical lip but a ridge of distinctly inflammatory or hyperplastic tissue. Its surface is engorged and often surmounted by edema of a cystic type or a more diffuse cobble-stone appearance, while often large flattened or pedunculated polyps are observed. The postmontane area is engorged and is vertical, or its rounded surface bulges toward the objective lense of the instrument; dilated prostatic ducts are often seen; the verumontanum is enlarged, shows surface edema, with ejaculatory duct openings evident at times. The findings indicating urinary obstruction, namely bas fond, trabeculation and hypertrophy of the trigone, are less in this prefibrotic stage, and residual urine is small in amount or absent. A moderate degree of intraurethral encroachment of one or both lateral prostatic lobes is not infrequently seen in these early types of cases; the encroachment is not marked and is seen to reduce and increase by turning the irrigating fluid on and off.

These inflammatory types of vesical neck changes present difficulties in diagnosis which demand the utmost judgment and care in differentiation on the part

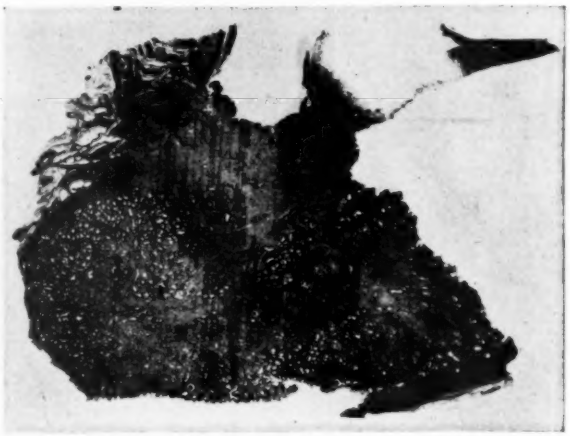

Fig. 7. Transverse section through vesical neck and adjoining prostate. Sphincter muscle shows extensive fibrosis with few compressed submucosal glands. Prostate shows definite but quiescent chronic inflammatory changes.

of the urologist. One can easily become a median bar enthusiast. The patient is often uncomfortable and frequently demands relief. The premature use of the punch or cutting current, in most instances, will not only be unsatisfactory, but results in a distinct increase in disturbance rather than an amelioration of symptoms. We believe the use of the punch should be reserved for those cases of quiescent fibrosis in which there is definite subjective and objective evidence of obstruction.

Has the frequency of occurrence of median bar been overestimated? We believe that while one frequently sees elevations of the posterior lip at the mortuary table and in routine cystoscopic examinations, they should not be confused with relatively infrequent, true fibrotic changes of the posterior lip for which the best treatment is removal by visualized punch or destruction by the cutting current, wherein the inflammatory process at the bladder neck, prostate and seminal vesicles has become quiescent.

We wish to express our appreciation to Dr. Charles Norris, Chief Medical Examiner of New York City, who permitted the use of necropsy material, without which this presentation would not have

been possible, and to Dr. Joseph McCarthy for the use of microscopic sections of some of his specimens removed by him with his visualized punch from median bars and also to Miss Alice E. Slavkin, technician, through whose untiring efforts and skill, thousands of excellent sections were obtained for this study. Our pathological work has been constantly supervised by Dr. Nicholas Alter and Dr. Lawrence Sophian, Pathologists at the New York Post-Graduate Hospital.

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DISCUSSION

Dr. Nicholas M. Alter: The pathologist is often confronted with great difficulties when trying to reconstruct the various stages of the pathological process. The findings in the autopsy room and in the operating room often show terminal or advanced stages of pathological lesions. This applies particularly well to the obstructive lesions of the vesical neck where probably the earliest stages of the process cause great functional disturbance without essentially interfering with the vitality of the patient.

Specimens removed at various stages of a pathological process lead to considerable confusion in the literature in regard to classification. Drs. Hyams and Kramer have undertaken an important and difficult task, trying to clear this confusion. In the various types of obstructions they see various stages of the same process. Drs. Hyams and Kramer were relatively conservative and modest in their conclusions considering the vast amount of work and material. I had opportunity to watch this work in my department. Drs. Kramer and Hyams have been studying about 2000 slides for a period of a year.

Dr. Lawrence Sophian: I have had the pleasure of going over the material that Dr. Hyams and Dr. Kramer selected. The two phases of the work which especially deserve to be brought up are: first, the soundness of applying general pathological concepts to special regions, such as the posterior urethra; the processes can be traced through acute, subacute, and chronic stages in the vesical neck. Second, the presence in that particular location of the small submucosal glands, which are not well understood, makes a problem in that the condition is likely to persist because such glands enable the bacteria to persist and reach a state of symbiosis. These glands are related embryologically to the prostate itself and have the same embryological derivation. They do not reach further than the superficial layers, but in some sections they could be traced further in; in others, they represented small pits. We hope that by having a larger amount of material the rôle which the superficial glands play can be worked out better at some future time.

ACUTE HEMORRHAGE FROM CORPUS LUTEUM AND GRAAFIAN FOLLICLE*

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ATLANTIC CITY, N. J.

Novack, in 1917, reviewed the literature concerning acute hemorrhage from corpus luteum and Graafian follicle and reported 39 cases of copious hemorrhage into the peritoneal cavity, due to rupture of either a Graafian follicle or corpus luteum, and reported 1 case of his own, bringing the total to 40 cases up to and including 1917. The literature apparently, has not been brought up to date since that time, although case reports on this very interesting entity have appeared from time to time. A recent patient, operated upon by the writer, prompted a desire to become more acquainted with this lesion and suggested that a review of the literature to date would be interesting and at the same time valuable and instructive.

Since 1917 there have appeared 36 additional cases[1] in the available literature, this case making 37 additional cases, since Novack's review or a total of 77 cases appearing in an exhaustive search. I am satisfied that many other cases have occurred that have either not been reported or have not been recognized as such. For instance, no reports have been found issuing from the countries of the Far East. Practically all the cases have been reported from North and South America and Western Europe.

The case report follows:

Case 1. Mrs. H. B., twenty-five years of age, white, married, was admitted to the Atlantic City Hospital April 7th, 1929. She had always enjoyed good health and had had no serious diseases. Until two years ago her menstruations were irregular, but her physician gave her a gland preparation and this was corrected. Married five years and has one child. She came to this city, with her husband, three days ago to celebrate their fifth wedding anniversary. She retired the evening before in perfect health and this morning, while in the act of coition, before voiding, she felt a severe pain over her symphysis and had to interrupt intercourse. This severe pain soon abated, but a continuous pain was present and she felt nauseated. I was called to see her about four hours later. Her temperature was 99°F., pulse 80, good color, entire abdomen moderately tender, but not rigid, the peritoneal rebound was very painful. Vaginal examination did not reveal any injury to the vaginal tract nor was there any softening of or bleeding from the cervix. A mass about the size of an English walnut was felt in the right iliac fossa. This mass was not excessively tender and was considered to be an ovarian cyst. The uterus had good mobility, but motion of it caused pain over the symphysis region. Menstruation was due in about three days and the last preceding period was apparently normal in all respects.

These findings were not definite enough to allow of a differentiation between ruptured ectopic pregnancy, twisted ovarian pedicle, intraperitoneal rupture of the urinary bladder, or a beginning pelvic peritonitis.

An enema gave no relief from pain and a catheter specimen of urine contained no blood. I saw her again about four hours later and the diagnosis of severe internal hemorrhage was evident at a glance. She was extremely pale, restless, asking for water, had sinking spells, pulse imperceptible at times and complained of pain about both shoulders and up the left side of her neck. A vaginal examination did not reveal any fullness or bogginess in the cul-de-sac and the abdominal examination was the same as before except that the right quadrant had become more tender. A diagnosis of ruptured ectopic pregnancy was made.

[1] Cases reported by Peuch, Corlette, Roggla, Burge, Cohn, Pobedinsky, Hadden, Wilson (2 cases), Duhail, Pen, Haggstrom, Rojas, Taylor, DePage, Gross, Speese, Orth, Moore (2 cases), Greenhill, Levi (3 cases), Wilson, Hammond & Stephenson, Hayden, Feiner (2 cases), Penny, Beall, Trousseau, Valliset, Scanzoni, Schumann, Ravdin.

* Submitted for publication March 25, 1930.

She was sent immediately to the operating-room and 750 c.c. normal saline was given intravenously before the pulse became perceptible and then nitrous oxide-oxygen anesthesia was given. The field of operation having been prepared and draped while the saline was being given, an infraumbilical midline incision was rapidly made and the mass in the right iliac fossa was delivered through a pool of bright red blood which was pouring out through the incision. This mass proved to be the right ovary showing a hematoma about the size of a pecan on its posterior surface and on the surface of the hematoma there was a laceration about ¾ in. long which was not bleeding because of traction on its suspensory ligament. The entire peritoneal cavity was apparently filled with red blood and there were a few very small blood clots. I estimate that there were between two and three pints of blood. The ovary containing the hematoma was removed and the adnexa on the left side examined and found normal. The appendix was found to be non-inflamed and was not removed. The incision was closed in layers without drainage and without attempting to remove the blood. A blood transfusion was considered, but the patient improved so rapidly that it was not necessary. She began to menstruate three days later. Her convalescence was uneventful and she was discharged two weeks later.

This case, and those included in this report, represent those serious intraperitoneal hemorrhages, which are in contrast to the rather common hemorrhagic cysts of the ovary, the latter including (1) ruptured Graafian follicles with slight bleeding, but definite hematoma, (2) hematomas of corpus luteum, (3) endometrial cysts, (4) hemorrhages into follicle cysts (5) and other types of ovarian cysts with hemorrhage into the cyst. In other words, this communication makes no attempt to include a study or classification of hemorrhagic accumulations within the ovary or even those showing moderate intrapelvic hemorrhage, but is devoted entirely to those cases causing a serious abdominal emergency and presenting the syndrome of acute internal hemorrhage.

I have been able to collect rather complete data on 25 such cases and have tabulated the outstanding points. This analysis has been very interesting.

Table I

Age	No. Cases
14–20	8
21–25	9
26–30	2
31–35	4
36–40	2

Table II

Relation to Menstruation	No. Cases
One week before	10
One week after	4
During	1
Two weeks after	3

Table III

	No. Cases
Graafian follicle	8
Corpus luteum	18

Table IV

Ovary Involved	No. Cases
Right	14
Left	10
Both	2

Table V

Preoperative Diagnosis	No. Cases
Appendicitis	13
Ruptured ectopic	10
Not stated	3

Reference to Table I shows that of 25 cases, in which the age was given, the majority have occurred before or during the

thirtieth year of life; 19 cases occurring before the thirtieth year while only 6 cases occurred after the thirtieth year. No case occurred before the beginning of menstrual life or after the menopause. The cases are about equally divided between married and single women.

Table II compiled from 18 cases in which the time relation to menstruation was stated, shows 15 cases occurring within the period of one week before, during, and one week after menstruation. Only 3 cases occurred in the remainder of the interval between menstruation.

Table III shows that 8 of the cases were due to a rupture of a Graafian follicle, while 18 were due to rupture of a corpus luteum. Therefore ruptured corpus luteum occurs about twice as often.

Table IV shows that the right ovary was involved in 14 instances, the left in 10 instances, the condition being bilateral in 2 cases.

Table V shows that the preoperative diagnosis was acute appendicitis in 13 instances and ruptured ectopic pregnancy in 10 instances.

Comparison of Tables IV and V suggests that the diagnosis of appendicitis is roughly equal in number to the cases showing hemorrhage from the right ovary, while the number of cases diagnosed as ruptured ectopic pregnancy corresponds to the number of ruptures of the left ovary. It therefore appears that when the right ovary ruptures and causes hemorrhage the condition is diagnosed as acute appendicitis, and when the left ovary is involved the diagnosis is usually ruptured ectopic.

So far as I have been able to determine the correct diagnosis has never been made before operation. Schumann reports 1 case in which he diagnosed intraperitoneal hemorrhage due to a ruptured follicle or corpus luteum and treated the case expectantly with recovery. The diagnosis in this case was not proved by operation and therefore the diagnosis must remain open to question.

Orth reports a very interesting case:

A woman twenty-six years of age was operated upon for acute appendicitis and a suppurative appendix removed. During the fourth night after operation the patient had uterine bleeding which was believed to be menstruation. On the following day, however, there was a striking and rapidly increasing anemia. A second operation was performed and 1.5 liters of blood was found in the peritoneal cavity. The source was a ruptured right Graafian follicle. After this operation the patient improved, but twenty-four hours later another hemorrhage occurred. A third operation revealed the source of bleeding to be a ruptured left Graafian follicle.

To discuss the etiology of profuse hemorrhage from a Graafian follicle or corpus luteum necessitates a review of the physiology of ovulation and the subsequent changes in the follicle after the ovum has been extruded.

ESSENTIAL PHYSIOLOGY

At birth the ovaries contain about 30,000 primitive or primordial follicles. Activity resulting in ripening of these follicles does not eventuate in the extrusion of the ovum until menstrual life begins. However, activity of growth probably begins even before birth of the child and this activity results in the formation of atretic follicles. After menstruation begins one Graafian follicle ripens and ruptures each month and many more never become ripe and go on to the formation of atretic follicles without extruding their contained ova. When, however, the Graafian follicle becomes ripe and the ovum is thrown out, rupture occurs through the stigma, hemorrhage of a physiological nature takes place and the cavity becomes filled with a hematoma. The formation of the corpus luteum then begins with repair of the laceration and organization of the blood-clot. It is thought that the cells lining the original Graafian follicle, the granulosa cells, enlarge and possibly multiply and that lutein is deposited in these cells, probably from the organizing blood-clot, giving the characteristic yellow color to the corpus luteum. The blood vessels,

which enter through the hilus of the ovary, give branches which supply each developing ovum and during the formation

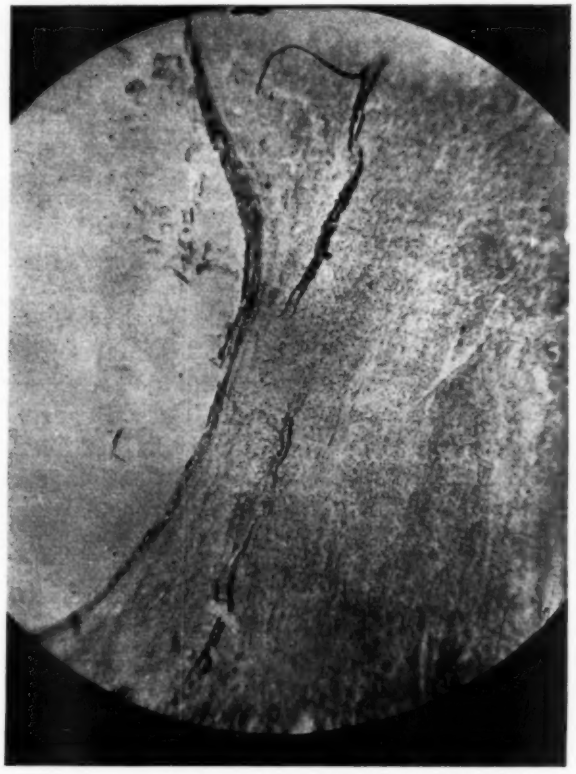

Fig. 1. Low power (*ca.* 75) showing edge of follicle into which hemorrhage occurred.

of the corpus luteum, in the lutein layer, are found many thin-walled vascular bud-like projections. The corpus luteum has a very short life, about three or four weeks, and this is followed by the corpus albicans which finally disappears, leaving a permanent scar on the surface of the ovary.

THEORY AS TO CAUSE OF HEMORRHAGE FROM CORPUS LUTEUM

We have seen above how thin-walled vascular bud-like projections are present in the lutein layer during the formation of the corpus luteum. It is easy to imagine injury or a solution of continuity of these fragile vessels brought on by a sudden hyperemia of the pelvic organs, as by sexual excitement, or by a sudden increase in the intravascular pressure due to sexual excitement or increased intra-abdominal pressure. After injury to these vessels a hematoma forms in the corpus luteum cavity and if hemorrhage persists long enough the pressure in that cavity will

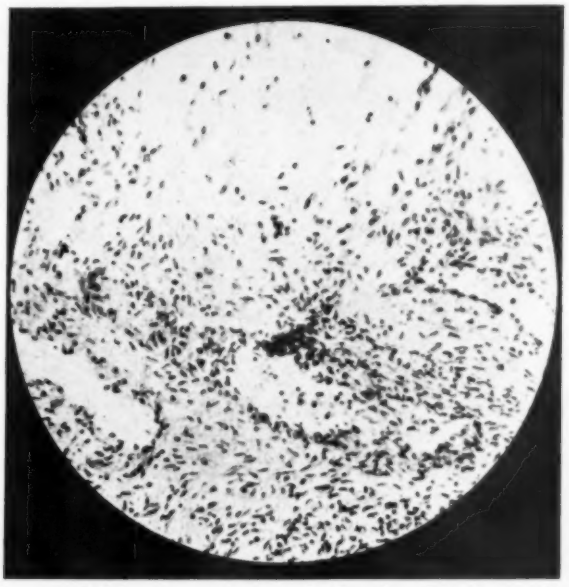

Fig. 2. High power (*ca.* 250) showing blood vessels without indication of pathological changes.

become so great as to burst the wall at its weakest point. It has been proved that a large proportion (60 to 80 per cent) of all ovaries removed at operation show hematoma formation in some part of their structure, showing the vulnerability of the smaller ovarian vessels. This bursting of the walls of the corpus luteum might eventuate in a copious hemorrhage or in a trivial one. This seems to me the theory most likely. Whether there is a pathological condition of the ovarian (or more strictly, thecal) vessels, as shown by Schumann to be present in his case, remains unsettled. Reference to the pathological study in my case is included in this paper. The pathological changes in the vessels in Schumann's case is a very interesting observation, but whether there was any relation between such pathology and the hemorrhage is debatable. The question of cause and effect is not thereby demonstrated for there is no reason why the hemorrhage could not have been a coincident condition. I do not believe that changes in the vessels alone would satis-

factorily explain the occurrence of hemorrhage.

THEORY AS TO CAUSE OF HEMORRHAGE FROM GRAAFIAN FOLLICLE

Returning to the normal physiology of the ripening Graafian follicle, we note that hemorrhage occurs into the cavity after the ovum has been expelled. This hemorrhage occurs from the thecal vessels. The additional physiologic hemorrhage from the lacreation through the stigma is no doubt negligible because the point of rupture has become so thinned out by the pressure of the developing follicle. A pressure ischemia of an obliterative nature would seem most logical in the production of the stigma. Presuming that in the ovary a Graafian follicle is almost, but not entirely ripe, when a sudden hyperemia of the pelvic organs occurs and causes an increased intravascular pressure in the ovarian blood vessels, premature separation of the ovum within the follicle might occur. This premature separation would produce hemorrhage into the follicle and if this hemorrhage was continued sufficiently long a bursting of the follicle at its weakest point would naturally occur. Intraperitoneal hemorrhage would then take place from both the thecal vessels and from the laceration in the ovary. This hemorrhage might be copious or trivial. I believe that in all the cases presenting serious hemorrhage such a process occurs in Graafian follicles that are not quite ready to rupture. In other words a premature separation of the ovum, with hemorrhage, and premature rupture of the follicle are the mechanisms producing the condition under discussion.

It is very significant that in the 25 cases, used as a basis of study for this paper, that increased intra-abdominal pressure was specifically mentioned in 9 cases. Eight of these patients stated that intra-abdominal pressure was increased either by vomiting or by straining at stools. In view of the frequent diagnosis of appendicitis in these cases and realizing that the majority of appendicitis cases vomit one or more times, I am satisfied that the incidence of increased intra-abdominal pressure would be greatly increased if definite records of this point had been made in all cases. It is interesting, however, that it was definitely stated in approximately one-third of the cases studied.

Dr. Robert Kilduffe, Director of Laboratories at the Atlantic City Hospital, has furnished the following report on my case after a very thorough microscopic study of the excised ovary.

Mrs. H. B. The specimen consists of an ovary which has been opened in its long axis. The tissue is pale in color and appears fibrotic. At one margin is a corpus luteum. In another area there are several punctate hemorrhages which appear to have arisen in the walls of a Graafian follicle.

Microscopy: (formalin fixation; paraffin sections.)

Particular attention attaches to the area in which hemorrhage has occurred. There is a partially organized blood-clot, the margin of which is enclosed by a layer suggesting in appearance the theca interna folliculi. The blood vessels throughout the sections show no evidence of pathological changes, which also are absent in the ovarian structure in general. There is nothing in the sections to indicate the cause of hemorrhage.

It is now interesting to note the rather positive value of this pathological report, in that there was no demonstrable disease of the ovarian vessels or specialized cells. It is thus demonstrated that in this case we began with an apparently normal ovary going through the physiological cycle of ovulation and maturation, that something suddenly went wrong, that a hematoma formed within the follicle apparently, and that later the follicle ruptured. This must evidently be called an accident. Increased intra-abdominal pressure and pelvic engorgement, both brought on by cohabitation, apparently was the exciting factor.

Novack writes:

The cause of hemorrhages emanating from the ovary (what might be called perforative ovarian hemorrhage) is in a large measure similar to those of ovarian hemorrhage of the non-perforative type, in which hemorrhage is confined in the ovary, giving rise to hematomas of various sizes. The principal predisposing factors, as might be expected, are conditions which bring about hyperemia of the ovary, with engorgement of its vessels. This hyperemia may be of either the active or the passive type. Practically always, as my study has shown, the bleeding comes from the thecal vessels of structures derived from Graafian follicles, but even more often from the walls of corpora lutea or corpus luteum cysts. The occurrence of free bleeding from the ovary into the abdominal cavity would thus depend largely upon the follicular structure involved. If it lies near the surface it can be easily understood that the thin wall may be broken through by the blood which escapes from the engorged vessels and free abdominal hemorrhage thereby results.

DIAGNOSIS

What is the possibility of making a correct diagnosis before operation? This study has found 77 cases reported with a diagnostic error of exactly 100 per cent, no case having been diagnosed correctly previous to operation.

The greater number of mistakes have been made in diagnosing the condition as acute appendicitis, because of the pain in the right lower quadrant, vomiting, fever and leucocytosis. It therefore appears that the physical examination must be the all important factor. The chief differences in favor of hemorrhage from the ovary would be:

1. Pallor of skin and mucous membranes.
2. More shock.
3. Fainting or sinking spells.
4. Less amount of rigidity of rectus which, in fact may be absent.
5. Area of exquisite tenderness is rather definite in appendicitis, while with ovarian hemorrhage the tenderness is rather diffuse over the lower quadrants.

The differentiation from ruptured ectopic pregnancy is more difficult. They both have in common:

1. Cramp-like pains over one or the other lower quadrant.
2. Fainting or sinking spells.
3. Evidence of internal bleeding.
4. Fever and pulse elevation.
5. Leucocytosis.

Ruptured ectopic pregnancy usually gives a history of one or more abnormal menstrual periods, there is usually some vaginal bleeding, softening of the uterus just proximal to the internal os may be present, and the breasts may show the changes of early pregnancy. If there is bleeding, not menstrual in type, from the uterus the case is probably not one of ovarian hemorrhage. This vaginal bleeding was present in only 1 of the 25 cases studied.

However, it is much easier to make the diagnosis on paper than it is in practice and without practical experience to keep this possibility in mind past records suggest that the condition will be confused with the two commoner ones, appendicitis and ectopic pregnancy. The diagnosis should not be missed by those who have encountered the condition.

TREATMENT

The treatment is immediate operation. A midline infraumbilical incision is the proper approach. Resection of the Graafian follicle with repair of the ovary is the operation of choice and should be done when the patient's condition permits. Removal of the entire ovary can usually be accomplished more rapidly and should be done without hesitancy if the patient is in poor condition. No drain should be used and neither should the blood be removed from the peritoneal cavity, although it is probably wise to remove large blood-clots. Examine the appendix but do not remove it unless the patient is in very good shape. Examine the opposite ovary as bilateral hemorrhage may be present.

PROGNOSIS

Of 77 cases appearing in the literature there have been 6 deaths reported.[1] This number is probably too small, but if correct, means a mortality of nearly 8 per cent. Two of the patients died within a few hours without operation.

SUMMARY

1. A review of the literature with report of an additional case is presented, making a total of 77 cases.

2. A critical study of 25 cases, in which rather complete data was available, has been attempted.

3. Acute hemorrhage apparently may occur from a normal ovary and present a serious abdominal emergency.

4. A theory as to etiology is discussed.

5. Diagnosis, prognosis and treatment are outlined.

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RENAL TUBERCULOSIS

CHANGING CONCEPTIONS IN THE DECADE 1920-1930*

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IN reviewing the literature on the subject that has appeared during the last decade one is impressed by the change in long-cherished ideas. The teaching that renal tuberculosis is never primary but always secondary has so assumed the dignity of age that it is seldom questioned, and when tuberculosis is found elsewhere in the body its rôle as a primary focus is at once cast and the renal involvement is considered secondary. The idea has rarely been considered that tuberculosis of the bones and joints, or other extrapulmonary tuberculosis, probably originates from the same focus that affects the kidney and that the renal involvement is not secondary to the tuberculosis of the spine or hip but coincident with it.

It seems probable, after careful consideration, that the original focus is usually in the respiratory system. A lymph node in the hilum of the lung becomes involved, the bacilli of tuberculosis readily pass to the lymphatic duct and thence to the blood stream; their final situation becomes a question of the relationship of local resistance to the amount and virulence of the infection present. If joint, bone and kidney are all involved, the involvement of the various structures probably occurs approximately at the same time.

If this hypothesis is correct, then involvement of both kidneys must be more common than our clinical experience would seem to indicate. This idea was formulated and presented, with clinical evidence, by Chute in 1920. He reviewed 3 cases which occurred in his practice and he considered that there were indications in occasional cases that tuberculous processes of the kidney healed. He pointed out how such a belief is necessary if one is to accept the hypothesis of the hematogenous route of the infection. He said:

> I believe we must assume that tubercule bacilli are brought in approximately equal numbers to both kidneys, but little cortical tuberculous infections are common and that far from going on to the complete destruction of every kidney that is infected they are probably promptly stamped out in by far the greater majority of instances. The generous blood supply of the kidney allows it to overcome perhaps the greater number of infections while yet they are incipient.

The wisdom of this hypothesis has been emphasized yearly in the last decade by clinical or experimental work, and we believe the evidence to be so conclusive as to convince urologists that earlier teachings of this subject need modification.

In 1921, Hyman and Mann reviewed 126 cases of renal tuberculosis observed at Mt. Sinai Hospital in the previous eight years. They concluded: "The presence of tubercle bacilli alone in smears or in catheterized ureteral specimens does not justify a diagnosis of renal tuberculosis; pus or microscopic blood must be found associated." This may be interpreted, although not so intended by the authors, in the light of work then being done by Lepper, as meaning that until the tuberculous process had extended so that blood and pus were present in the urine, the diagnosis of renal tuberculosis was not justified, because such a diagnosis implied the necessity of nephrectomy. While Hyman and Mann were collecting their clinical data, Lepper was investigating the experimental production of infections of the urinary tract with coliform organisms. She found, with but a single exception, that after the intravenous injection of coliform organisms there was always evidence of renal changes before the organisms appeared in the urine.

In 1922, Dyke presented a paper in

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which he demonstrated that cocci cannot pass through a normal kidney, and he threw grave doubt on the theory that secretory bacilluria of any kind can take place. In reviewing the literature, he found that Cohnhein, in 1882, apparently without making any experimental observations, first expressed the belief that bacteria pass through the normal kidney. The first experimental work was carried on four years later by Wyssokowitsch who concluded that the appearance of bacteria in the urine is dependent on the initiation of lesions in the substance of the kidney for he could not demonstrate urinary infection for at least six hours after intravenous injection and then only when the infection was associated with the presence of blood in the urine. His observations were confirmed by Boccardi, working with Banthrasis in 1888, and Pernice and Scagliosi, in 1892, using staphylococcus, concluded that organisms do not pass through an uninjured kidney. Sherrington, in 1893, reinvestigated the problem with the utmost thoroughness, and stated, "At a time when the blood is teeming with microörganisms there may not be the slightest transit of them into the urinary fluid then secreted." These data were further confirmed by Sittman, in 1894, and by Colten, in 1895, who demonstrated injury of the kidneys which had proved permeable to the passage of organisms, and he concluded that they were excreted only when lesions were present in the kidney.

Investigators up to this time had been in agreement and had employed a technic which usually consisted of obtaining specimens of urine without the use of catheters by puncture of the bladder with small needles through seared surfaces.

Biedl and Kraus and von Klechi, in 1896, investigated the subject. They placed cannulas in the cut ends of ureters and found that the kidneys excreted the organisms almost at once. As a result of their work the theory of secretory bacilluria became well established and it was not until the work of Dyke and Lepper, and more recently of Helmholz, that their error in technic was demonstrated and that the more extensive and accurate work of the earlier investigators was again established. One can now readily appreciate how subject to contamination must be any urine obtained by a technic that involves cutting of the ureters when the entire circulatory system is filled with bacteria. Thus, a slight error of technic established an erroneus teaching.

In 1923, an incident took place which led to an important further study. A surgeon performed nephrectomy because bacilli of tuberculosis were found in a specimen of urine from the ureter. Since gross pathological changes could not be demonstrated in the removed organ, the surgeon attributed the pathologist's finding of the organisms to an excretory bacilluria and threw on the pathologist the responsibility for removal of the evidently normal kidney.

The pathologist was Medlar, and late in 1924 he and Sasano, after a most careful experimental investigation, published their work entitled "Experimental Renal Tuberculosis with Special Reference to Excretory Bacilluria." After intravenous injections of bacilli of tuberculosis had been given to a series of guinea pigs, their urines were injected into other pigs, after which the kidneys of the pigs with urines positive for acid-fast bacilli were examined. Medlar found that it was necessary to run serial sections in order not to overlook lesions, but he found, by this method, that every pig with a urine positive for acid-fast bacilli had renal involvement. He concluded that in the numerous cases reported in the literature in which organisms of tuberculosis have been reported to have been excreted from normal kidneys, lesions exist which only serial section will demonstrate.

In examining serial sections, Medlar found lesions that were in the process of healing and he concluded his report with the assertion: "Individual tuberculous

lesions of the kidney may heal," and further, "No evidence to support the excretion of tubercle bacilli from normal kidney has been found." He also observed that experimentally the size of the tuberculous lesion is no criterion as to the number of bacilli present and that there may be extensive renal involvement before any organisms get into the urine.

Also in 1924, Bugbee reported 2 cases representing unusual types of renal tuberculosis in which bacilli of tuberculosis could not be isolated from the urine or demonstrated by inoculation of guinea pigs. He concluded his article with the following sentence: "They lead one to believe that tuberculosis of the kidney probably exists much more frequently than is generally conceded, that a correct diagnosis of this type of case is often overlooked, and that such a low grade tuberculous renal infection is self limiting in some cases." Thus was clinical evidence presented to substantiate Medlar's experimental work. The latter presented his experiments before the American Association of Genito-Urinary Surgeons and in the discussion of the paper Hagner remarked:

> Another point is the occurrence of the tubercle bacilli in the urine without tuberculosis in that kidney. Experimentally that may occur but I would like to ask any man in this audience if he has ever seen a case in a human where tubercle bacilli were present in the urine, operation done, and no tuberculosis found in the kidney. I have never seen such a case. If we find tubercle bacilli in the urine, I believe that patient has tuberculosis of the kidney.

This illustrates how rapidly the ideas of renal tuberculosis were changing even by the middle of the decade.

In June, 1925, Helmholz and his associates published the first of a series of papers entitled "The Kidney, a Filter for Bacteria." They did not find bacteria in the urine until sufficient time had elapsed to permit of renal injury and they were always able to demonstrate the injury microscopically. They used many different bacteria, and even produced extensive lesions with one organism; they then injected a different organism to see if it would come through at once, and in one series of experiments they produced hydronephrosis before the injection. But even in the presence of gross pathologic change, the kidney held back the organisms sufficiently long for characteristic lesions to form. Helmholz remarked:

> The generally accepted mode of sterilization of the blood is not by excretion but through phagocytosis by the endothelial cells and in the endothelial cells of its large capillary bed. The kidney possesses a large phagocytic field which can dispose of large numbers of bacteria. The kidney can and does rid the circulating blood of bacteria but by means corresponding to those of other organs and not by secretion of organisms.

The next year, in 1926, Medlar reported on the pathologic examination of 100,000 serial sections from the kidneys of 30 patients who had died of advanced pulmonary tuberculosis but who had not had clinical symptoms of renal involvement. In 22 of these cases, renal tuberculosis was found. In every case in which both kidneys were examined, the disease was bilateral. Since definite scars were present in 17 of the cases, he concluded that tuberculous lesions of the kidney heal.

Previously Braasch and Scholl had reviewed 22 cases of renal tuberculosis in which nephrectomy had been done and guinea pigs had been given injections of urine from the supposedly normal kidney. They were surprised to find evidence of tuberculosis in 7 cases. Beer had reported similar results and had attributed them to renal reflux.

We recently have reviewed the ultimate results in cases seen at The Mayo Clinic up to January 1, 1929, in which guinea pigs were given inoculations with urine obtained before operation from the supposedly normal kidney (Table 1). There were 175 such cases; in 23 the test was a failure, in 109 negative results were obtained in the guinea pigs and in 43 the

results were positive. Two of the 43 patients died in the hospital and 11 died subsequently. Of the 30 who remain 13 have unquestionable involvement of the remaining kidney; 3, we could not trace, and 14 are well. Urine from 2 of these when inoculated into guinea pigs, in the course of reexamination of the patients, has given negative results. Do these 14 cases represent instances of reflux of urine up the ureter of the good kidney at the time of examination, as suggested by Beer, or do they represent early involvement of the kidney that subsequently has healed? Possibly they represent a proportion of both. It surely seems somewhat dogmatic to assert that, if the patient dies subsequently, that the ureteral specimen gave a correct impression of disease of the kidney, whereas, if he lives, the interpretation must always be that there has been an error of technic. For after the splendid investigative work of Helmholz, Lepper and Dyke one can no longer advance the conception of excretory bacilluria as an explanation.

Also in 1926, Morse and Braasch, in their article on the comparative value of inoculation of guinea pigs in the diagnosis of renal tuberculosis stated; "The clinic records do not show a single case in which the diagnosis of renal tuberculosis was made from finding a positive stain or guinea pig inoculation in which tuberculosis of the kidney was not found at operation."

In 1927, Thomas and Kinsella gave their preliminary report of clinical research in renal tuberculosis that they had carried on for a number of years at Glen Lake Sanatorium in Minnesota. Their results were strikingly similar to those which Medlar obtained. They wrote:

> We cannot understand why tubercle bacilli behave differently in the kidney than in other tissues in that a renal lesion is not supposed to heal. We believe that tubercle bacilli are carried to the kidney by the blood stream and that every renal infection with tubercle bacilli must be primarily bilateral. One kidney may not overcome the infection as well as its mate and a destructive lesion develops. We have never removed a kidney which eliminated tubercle bacilli that did not contain a lesion of tuberculosis. Renal tuberculosis may end in clinical arrest or cure.

In June, 1928, Wildbolz of Switzerland delivered in Chicago an address on renal tuberculosis. He attributed the recovery of approximately 60 per cent of his patients on whom operation had been performed to the fact that renal tuberculosis was usually unilateral, but he refused to attribute the 40 per cent of deaths to the possibility that the disease ever originated as a bilateral infection. He energetically expressed his disapproval of the radical views which were developing in America concerning the etiology and progress of the infection, and he held firmly to the long-taught theories of secretory bacilluria and inability of a tuberculous renal lesion ever to heal. The chief interest of his paper lay in his description of tuberculous nephritis; he said:

> Caseation and cavity formation are lacking in the infected tissue but there is also no formation of tubercles. The renal parenchyma does not react in such cases to the invasion of the tubercle bacillus with specific tuberculous tissue changes; on the contrary the bacilli may not infrequently produce only a non-specific infiltration with a tendency to fibrosis of the kidney. Why the tubercle bacillus in rare instances causes only non-specific inflammatory changes in tissues without tubercle formation or caseation cannot be stated with certainty.

The lesions he described resembled somewhat those described by Bugbee in 1924, and Wildbolz explained some of his apparent cures of renal tuberculosis to this type of the disease.

Also in 1928, we reviewed the clinical material relative to tuberculosis of the genital tract among the patients who had come to The Mayo Clinic prior to January, 1923. The cases which had occurred in the previous five years were excluded in order that the final results might be better

determined. In a review of 300 cases we found that dysuria was usually a symptom of urinary tuberculosis and that it occurred rarely when the disease was confined to the genital tract. We also found that "the presence of the bacilli of tuberculosis in the urine indicated renal involvement." In using the word "indicated" we intended to imply the idea of "probability." For instance, when the sky is overcast, one may say "that indicates rain." Doubtless it would have been clearer if we had said "almost always indicated renal involvement," for the sentence seems generally to have been interpreted as an absolute statement. At the meeting of this Academy last year, Beer referred to our conclusion as rash and he further stated: "In males, prostatic tuberculosis may lead to a clinical picture quite similar to the cystitis of renal tuberculosis and . . . perhaps 15 per cent may have tubercle bacilli in the bladder urine."

In spite of our high regard for Beer we believe it is only rarely that tuberculosis of the genital tract can be given as the source of bacilli of tuberculosis in the urine. Accordingly we have recently reviewed again the 300 cases of genital tuberculosis seen at The Mayo Clinic prior to 1923 and have included 306 additional cases examined prior to January, 1929. This series, which includes a total of 606 patients, we believe to be sufficiently large to enable accurate conclusions to be drawn. In 73 of these patients renal tuberculosis was proved to be present although the urine was negative for bacilli of tuberculosis. Two hundred and fifty-seven patients had urine that was positive for bacilli of tuberculosis: 170 of these patients underwent nephrectomy for renal tuberculosis. This leaves 87 patients of the 257 who were not operated on. Fifty-seven of these 87 patients who were not operated on have died since. In these 57 cases the renal source of the bacilli had been demonstrated by cystoscopic examination in all but 12, in which cystoscopic demonstration was not justifiable because the disease had advanced so far that renal calcification had resulted. Of the 30 cases which represent the difference between 87 and 57 the disease had invaded both kidneys in 20 and was unilateral in 8; this was demonstrated by cystoscopy. In the remaining 2 cases of the 30, that is, in 2 cases of 257 we were unable to demonstrate the renal origin of the bacilli of tuberculosis and we believe, as Dr. Beer has suggested, that the organisms may have been of genital origin. An incidence of less than 1 per cent surely will justify our saying that the presence of bacilli of tuberculosis in the urine almost always indicates renal involvement.

The last year of the decade we are reviewing was ushered in with an article by Harris entitled "Tuberculous Bacilluria." In scope and thoroughness of the work represented by this article, it has few equals. Its author studied 43 adults and 67 children who had surgical tuberculosis outside the urinary tract and found an incidence of 37 per cent of tuberculous bacilluria in adults and 13.8 per cent in children. In the majority of these cases, urinary symptoms were not present. We do not believe the frequency with which urinary tuberculosis occurs without symptoms is generally appreciated. In a recent review of 345 cases in which stained smears of urine contained acid-fast bacilli at The Mayo Clinic 28 patients (8 per cent) had no complaint referable to the urinary tract; from 23 of these patients, tuberculous kidneys were removed. The other 5 also had renal tuberculosis but operation was thought inadvisable.

Harris was interested to see if he could demonstrate that any of the unexpected incidence of symptomless tuberculous bacilluria was of the secretory type. At the suggestion of Klotz, he collected urine daily for ten days from 3 patients who were free of symptoms referable to the urinary tract but who had tuberculosis elsewhere. He wrote:

> Each of these daily specimens produced tuberculosis when inoculated into guinea

pigs. In other words, tubercle bacilli were present in the urine every day though the patients were free from symptoms. Were their presence due to excretion from the blood tream by the kidney, an equally constant tuberculous septicemia must have been present. A tuberculous septicemia so constant and severe could hardly exist without the occurrence of miliary tuberculosis. None of the patients had at that time nor have they at present any evidence of miliary tuberculosis. Blood cultures taken during the periods when urine was collected were free from tubercle bacilli. It seems certain, therefore, that the tubercle bacilli came from foci of renal tuberculosis.

At the conclusion of Harris' paper he added: "It is reasonably certain that the presence of tubercle bacilli in the kidney urine means the presence of tuberculous lesions in the kidney." Since a goodly proportion of the patients without symptoms, but with bacilli of tuberculosis in the urine, ultimately became free of the organisms, as indicated both by examination of stained smears and by the results of inoculation of guinea pigs, it is not strange that in his conclusions he stated: "The initial renal lesions frequently heal, less frequently they steadily progress to complete destruction of the kidney and death of the patient."

In June, 1929, Thomas brought to a close the list of stimulating papers on the subject of renal tuberculosis that had appeared in the decade. His was a report of a ten years' clinical study at the Glen Lake Sanatorium in Minnesota. Among his conclusions were two statements that ten years before would have been considered rank heresy: (1) "renal tuberculosis is in the majority of cases a bilateral condition" and (2) "non-destructive lesions will heal and the patient with this type of lesion should have the advantage of sanatorium treatment to assist him in building up a resistance against tuberculosis."

We do not feel that a review such as this, of the last ten years' work, is justified unless we can add some evidence that will contribute toward the change to the present conception of renal tuberculosis. Since many cases of extensive tuberculosis of the bones, joints and lungs, as well as many cases of tuberculosis of the urinary tract are seen at The Mayo Clinic, we hoped, by reviewing all cases in which there were bacilli of tuberculosis in the urine, to discover at least some confirmatory clinical evidence to substantiate the newer ideas relative to renal tuberculosis.

During the six years between 1923 and 1929, 5971 smears of urine were stained, of which 570 were positive for organisms of tuberculosis and represented specimens from 345 different patients. Two hundred and forty-three of these patients underwent nephrectomy for tuberculous kidney, either at the clinic or elsewhere (Table 11). The remaining 102 fell into 5 groups when studied carefully. In the first group were 17 cases in which the concentration of urea in the blood was so high as to indicate hopeless renal impairment, presumably bilateral; 13 of these patients are known to be dead. The second group of 47 patients, of whom 25 are dead, represents an earlier stage of the same condition; involvement was demonstrated as bilateral by cystoscopic examination, instead of being inferred from the demonstrated renal insufficiency. Some of these patients possibly might have been benefited by removal of the more involved kidney. At least, operation might have done no harm, for Braasch called attention to the fact that when nephrectomy had been performed, and, following this, urine from the supposedly normal kidney was found to have given a positive test on inoculation of a guinea pig, the subsequent mortality and the subsequent improvement are practically the same as in those with, so far as can be determined, strictly unilateral involvement. A third group of 20 cases comprised those patients with tuberculosis of the lungs or bones so extensive as to preclude any thought of renal surgery. Eleven of these patients are known to be dead. Of the remaining 9, 4 could not be traced, and 2 have persistence of vesical

symptoms. The urine of 1 of the other 3 patients, a man aged twenty-one years, was found to be positive for bacilli of tuberculosis. The left kidney was found to be the source of these organisms, and since all other observations gave negative results, heliotherapy was advised. The patient now reports himself to be well. The second patient was a man, aged twenty-four years with Pott's disease and pleurisy, without urinary symptoms or demonstrable genital involvement but whose urine from the left kidney was positive for bacilli of tuberculosis. Because of the multiple foci of tuberculosis, nephrectomy was not done and heliotherapy was advised. On inquiry three years later, he reported that he was doing manual labor in a section gang and feeling fine. The third patient, a child with Pott's disease, still is under treatment in a sanatorium. Possibly this was a case of excretory bacilluria, but after Harris' excellent investigation of this type of case, we are inclined to doubt it. The fourth group comprises 6 cases in which nephrectomy was advised but refused.

The fifth group comprises the 12 remaining cases out of the original 345 in which the source of the tuberculous bacilluria is in doubt. In 1 of these, calcification of the kidney, several years later, indicated the origin. In 2 others there was extensive genital involvement, and cystoscopic examination was not made. Since the pyuria was extreme, it is doubtful if the bacilluria was entirely due to genital contamination. One case had all the features of secretory bacilluria. Following the removal of a tuberculous tonsil, acid-fast organisms were found in a single specimen of urine. Subsequent specimens were all negative for bacilli of tuberculosis. Since 5 of the remaining 8 patients were women, we believe that the smegma bacillus was mistaken for the bacillus of tuberculosis. If, however, we accept the 7 cases in males, in this group, as examples of tuberculous bacilluria of genital origin, the proportion is only 2 per cent. However, in 4 of the 7 there was no palpable evidence of genital disease. Since tests with guinea pigs gave negative results in the 4 cases in which no evidence of genital disease was found, we believe the organisms found in the stained smear were probably smegma bacilli. As previously stated, 1 of the remaining 3 male patients was later found to have a calcified kidney, leaving but 2 patients who did not present evidence of a renal origin of the bacilli.

CONCLUSIONS

1. Initial tuberculous lesions in the kidney frequently heal.

2. It is not possible for a normal kidney to filter bacilli of tuberculosis out of the blood stream into the urine.

3. The presence of bacilli of tuberculosis in the urine almost always indicates renal involvement.

4. Dysuria is a symptom of urinary tuberculosis and does not occur when the disease is confined to the genital tract.

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EVALUATION OF DENTAL ROENTGENOGRAPHIC SHADOWS*

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THERE are two chief sources of possible error, or rather, differences in interpretation, in any roentgenogram. The first is the film, which of course is a variable factor because of the technic of the operator, and the second is the personal one. With the improved methods of portrayal of anatomical parts and more particularly the dental areas, and the modern perfected machines for roentgenography, a more standardized way of portrayal has been developed. Therefore, the exposure and development of the films, which is the first factor, will not be stressed but rather the second, the value to be placed on the shadows developed. Their easy identification as either expression of normal entities or of some pathological significance will be given greater consideration. It is presupposed that the technical procedure of exposing films of the dental areas will have been done in the most approved fashion. This must be done with a knowledge of the anatomy of the jaws, so that the bony landmarks will be portrayed in a manner that they may be recognized and prove of diagnostic assistance where necessary, and with sufficient bony definition to portray the intimate character of the surrounding alveolar process. It is in these areas that most of the changes of diagnostic value are to be found.

Assuming that the character of the film is such as to portray the teeth and their surrounding alveolar process in good contrast, we may then proceed to describe a rule from which we may formulate the value of the various shadows and their relation to the normal or abnormal or even pathological arrangement of parts.

It will perhaps not be amiss to call attention to the fact that the shadows or silhouettes seen on the film are not those commonly considered, but rather silhouettes within shadows. The appearance of the silhouetted parts is due entirely to the peculiarity of the roentgen-ray force: the ability of penetrating areas in proportion with resistance encountered. We record this invisible penetration on the sensitized film. The force of this penetration is registered on the film in inverse ratio to the resistance encountered, and is recorded because of reduction of the silver coating in direct ratio to the force delivered. The resistance is variable, and in the anatomical areas is dependent on the inorganic salt composition of the parts, the atomic weight of these salts and the density of their formation. A review of any analytical chemistry will indicate that there is a rather wide range of difference in the inorganic salts component of anatomical tissue. Bone, as indicated by the examination of the ash residue, has a 61.50 per cent inorganic residue, as against 0.88 per cent for the soft tissues. Of the two chief inorganic salt components of this residue, calcium oxide exists to about 28.55 per cent and phosphoric oxide approximately 19.55 per cent. The atomic weight of calcium is approximately 40.07 and that of phosphorus is 31.04, as indicated by the international standards of atomic weights of 1918. The difference of resistance, therefore, as it exists in these two chief grades of tissue, soft tissue and bone, as computed by the difference in the atomic weight of their two chief component salts is as approximately 1 to 70. Considering the density of the shadow representing that tissue with a minimum of inorganic

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salts as the indication of ray force penetration, it is 70 times that indicated in an area representing bone or with reference to the dental areas, the teeth, where we find the greatest concentration of inorganic salts of high atomic weights.

Fig. 1. Fig. 2. Fig. 3.

Fig. 1. Two anatomical classes of shadow. From outline of finger-tip to outline of bony phalanx, smooth gray shadow area represents soft tissue with minimum of inorganic salt content, termed Grade I shadow. Within the heavier shadow outline representing phalanx there is another gray shadow, deeper in tone and rather mottled. Represents cancellous bone of Grade II a value. The two teeth are heavier gray or almost black, representation of heavier bone, and indicate Grade II b shadow. (Note: Shades reversed in negative.)

Fig. 2. Pulp chamber and root canals visible through tooth and bone structures. Note difference in outline of various pulp chambers. Pulp in first molar is putrid and because of lack of circulation in it, it is pictured sharply outlined in contrast to others, with circulation, which are in less sharp definition. Note filling (Grade III shadow) in second bicuspid with overhanging margin distally. Some Grade I shadow value to be seen between this filling and shadow of tooth structure. Indicates destructive process and in this instance decay. (Findings were shown to be correct after having been made originally from negative.)

Fig. 3. Normal distribution of Grades I and II shadow in jaw of a ten year old child with deciduous molar and unerupted bicuspid under it, Note Grade I shadow about crown of erupting tooth, which is a portrayal of tooth capsule of soft tissue. Note same character shadow about unformed root of erupting tooth, and similarly only to lesser degree about erupted bicuspid, which is not yet fully formed.

In the area recorded as soft tissue there is little definition and little change in the character of shadow possible except that of contour. A destructive process which would remove some of the inorganic salt content would allow the increase of force penetration to register to such an extent. This would be insufficient to develop any visible contrast. Because the density of the reduced silver coating in such a film area, a slight increase is harder to define than a corresponding decrease would be. A process however, of construction, growth, or deposit of greater inorganic salt content, if within the area of greater penetrability, becomes more easily definable. Note how easily the gingival ridge is portrayed in the edentulous jaw, and particularly so if this ridge is dense and fibrosed. Too, the wooden blocks, so often used to hold the dental film in place, can be defined and outlined over an area of gingival shadow, even compounded through the cheek. Illustrations of this character are an indication of the increasing of resistance to penetration and varying the density of the film coating to a visible ratio.

To give a name and a classification to this type of shadow, I have termed those film densities which portray the soft tissues as Grade I shadows. This is in contradistinction to the shadow of bone parts with their greater inorganic salt content and greater resistance to roentgen-ray penetration, which is classified as Grade II shadow. It is obvious that there are two types of bone portrayed, the loose alveolar type or cancellous bone seen about the jaws, and in which the roots of the teeth are imbedded, which can be defined as Grade II a, and the densely compacted bone as represented by the teeth and the bone cortex, under the heavy periosteum, which may be termed Grade II b.

A study of the shadows thus classified would quickly indicate the anatomical tissue in which the portrayed and observed is generic. The anatomical position of any type of shadow is fixed. Its position and extent must be governed by the anatomical area portrayed, and so must

the ratios, therefore, of Grade I and II shadow correspond to the ratio of their normal existance in the area. Deviations must be recognized as the representation of the extent of any change taking place. The value of the alteration as a pathological entity must then be determined.

Without any history or clinical data, it falls upon the dental roentgenologist to determine first if there is or is not a lesion in the areas he is observing. Such determinations depend primarily upon experience, for without it he would not be able to recognize a normal portrayal of the parts and a proper ratio of the two chief grades of shadows. The intimate contact of the intraoral film with the anatomical part portrayed permits of the outline of the area in rather exact detail. This detail is so correct that it is possible to portray minor changes which take place with development, growth, and even such that occur within the tooth structure as the result of an alteration in circulation. It is obvious when referring to growth, that reference is not being made to deciduous teeth, but to growing teeth of the second dentition. Carefully made dental films as carefully read, should indicate such changes in tooth development as are compatible with growth, and differentiate such as are the result of some pathological process. These determinations are based on the known rate of development of teeth and the closing of the apical end of the root canals, as well as a knowledge of the size and shape of pulp chambers. It is an observation and interpretation made by virtue of the recognition of the normal ratio of Grade I and Grade II shadows.

In the dental areas there are often fillings in teeth and in root canals, which under certain conditions have no pathological significance, and at other times are the site or origin of the condition complained of. The majority of this reparative work is done with metals or metallic salts of high atomic weight. Their resistance coefficient is for the most part greater than is that of dense bone. It is therefore necessary to consider a third type of shadow for this area. Grade III type of shadow embraces that of all character of filling material and is normally found within the tooth or within the root canal as filling material, or a replacement for a decayed portion of tooth. As a replacement, the filling material should restore the normal contour and the size of the tooth.

Each grade of shadow defined as visible in the dental area has its individual characteristics. Each has its anatomical position in which its portrayal will indicate its normal or pathological extent. With improper portrayal, each may be distorted. It is therefore necessary to understand the anatomy of the teeth and jaws, the variations possible within the normal limits and the changes due to normal development of the parts, or their alteration due to repair.

It is axiomatic that the finding of a shadow typical of a certain grade, in the position or of the shape normally that of another, calls for an explanation of the condition as either a normal process or a pathological lesion. Likewise, the alteration of the shape of a shadow in any grade of tissue, from that recognized as normal, must stand the question of its being a normal or abnormal one. To detail what type of lesion is responsible for a definite train of symptoms would require too great amount of space and too much time, but suffice it to say that the acute lesion is represented in Grade I type shadow, and is noted as an increase in the normal ratio of this grade of shadow over Grade II. Destructive lesions, such as infections, abscesses and cysts fall into this class. If one considers a lesion in bone (Grade II shadow), however, one has to deal with the destructive or osteoclastic lesion, where the process may be defined or identified as a replacement of Grade II shadow by Grade I or the proliferative or osteoclastic type of lesion in which Grade II shadow is made more dense. Osteoclastic lesions are usually found in the cancellous bone

areas, and here it will be noted that the shadow defined as Grade II a is assuming the density and characteristics of that of Grade II b. In shadow grades, this would be described as the displacing of Grade II a shadow by an increase in the extent of Grade II b for the normal ratio for the area. The chronic lesion is most often pictured as one indicating greater resistance to roentgenographic penetration. The 1 to 70 ratio in the soft tissue shadow is shortened, and in Grade II shadow the sharp normal outline characteristic of the parts is lost.

In this paper there has been no attempt made to point out characteristic lesions, or to apply the rule of grading shadows here indicated, because of space. To give one's personal opinion as to the pathological significance of any finding makes for unnecessary controversy; therefore it will not be attempted. Suffice it to say that along with the recognition of shadows as to grade type and the placing of a resistance value on the reduced emulsion of the film, one must necessarily place a corresponding value in inorganic salt or mineral composition for the area. It is the determination of these as a correct and normal ratio which forms a basis for the determination of the possibility of a lesion and its pathology.

CASE REPORTS

OPERATIVE REPAIR OF THE ANTERIOR CRUCIAL LIGAMENT OF THE KNEE-JOINT*

ARTHUR KRIDA, M.D., F.A.C.S.

NEW YORK

THE symptoms of rupture or of attenuation of the crucial ligaments are primarily those of instability of the knee-joint. This may be a circumscribed instability referable to one or the other crucial ligaments. If the anterior ligament is frankly torn, there is increased anteroposterior mobility in extension; if the posterior, there is increased mobility backwards of the tibia on the femur, in flexion.

The classical circumscribed instability as outlined here and as originally described by Hey-Groves,[1] is not commonly seen. In my series of 8 cases, general knee-joint instability was pronounced in all but one, and the characteristic crucial instability was only a part of the general picture. In 2 of these cases, there was a frank rupture of the anterior crucial ligament; in 3 others, both ligaments were represented by very thin fibrous bands; in the remaining 3, marked attenuation and relaxation without rupture were encountered.

I conclude therefore that any knee-joint presenting the symptoms of chronic gross instability is likely to have crucial ligament damage, and that operative procedures which leave this out of account are not likely to be successful.

There can be no doubt that the crucial ligaments are torn in all complete dislocations of the knee, also that complete tears occur from lesser degrees of violence. The torn ligaments do not regenerate. Despite this fact, many such joints recover with good active but poor passive stability, The active stability is supplied by the quadriceps extensor muscle and while the muscle maintains a high degree of efficiency, such joints are perfectly useful joints. Such cases are naturally not submitted to operation.

In this series, all patients gave a history of a moderately severe wrench of the knee-joint. In none was there a history of complete dislocation. All complained of pain. Several could not walk without a brace; others could get about without support, but had recurring falls due to giving way of the joint, and such cases had recurrent effusions. As already stated, all but one of these joints presented general gross passive instability with impaired active stability; the remaining case, reported later on, had good lateral stability, but had the characteristic circumscribed anterior mobility.

The technic of operation was described in a previous paper.[2] It is a combination of the Hey-Groves and the Alwyn Smith operation, but instead of the curved incision across the patella tendon, I prefer to open the joint medially by the incision I have described as the general utility incision.[3] A second incision is made on the outer side of the thigh, a long strip of fascia lata is turned down from above, remaining attached below. A hole is drilled through the external condyle of the femur into the intercondylar space, and a second one through the internal tuberosity of the tibia to in front of the tibial spine. The fascial strip is drawn through the drill holes from above downward, is sutured below the periosteum of the tibia, and the

* Presented before the Section of Orthopedic Surgery, New York Academy of Medicine, February, 1930.

redundant portion of the strip is reflected upward to the internal condyle of the femur, thus reconstructing an internal lateral ligament.

Of the 8 cases, 6 have resulted in painless, stable knees with a full range of motion; one could be followed only ten weeks after operation, and had a satisfactory result for that time period; 1 was a failure, in that the disability was not relieved.

CASE PRESENTATIONS

(Three operated cases were demonstrated, the first having been operated upon in October, 1924, and reported in the paper previously referred to.[2])

Case II. S. F., aged eighteen, Hospital for Ruptured and Crippled, First Division. Twisted left knee three months previously. Recurring giving way and effusion since that time. Exploratory operation, February 19, 1927. Complete rupture anterior crucial ligament; an incomplete shelf across the quadriceps pouch, ending in a falciform edge.

Operation: reconstruction of the anterior crucial ligament by the technic described already; excision of the fibrous band in the pouch.

Discharged from hospital on the twenty-sixth day, walking with the aid of a stick. Full motion gradually returned without treatment. When seen in February 1930, he stated that he had had no further trouble, and that he had at various times played football.

Case III. F. L., Aged twenty-six, U. S. Marine Hospital No. 43. One year previously slipped on wet deck and struck right knee. Immediately disabled. In hospital four months, during which stay the joint was aspirated, followed by physiotherapy. Went back to work for four months, but the knee was weak and unstable. It gave way again, and he was readmitted to hospital, where he had another period of physiotherapy, when he was seen by me after a four months' stay. The quadriceps was markedly atrophied, and there was general laxity of the joint including abnormal anterior mobility of the tibia on the femur.

Operation: exploration of knee December 5, 1929. Greatly relaxed anterior crucial ligament; adhesions between fat pad and head of tibia. Excision adhesions, reconstruction of anterior crucial and internal lateral ligament. After four weeks, walking with the aid of a stick. In February 1930, about seventy-five days after operation, the knee was painless and stable, he had flexion to 90° and could go up and down stairs with ease.

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DISCUSSION

Dr. Armitage Whitman: I wish to emphasize the point made by Dr. Krida, that even after the most successful reconstruction operation, as has been demonstrated by the examination of these cases, there is still abnormal anteroposterior mobility of the tibia upon the femur. The cases he has shown, however, have such well developed quadriceps muscles that they can compensate for the ligamentous instability. I think we should all do well to remember that stability of the knee-joint is dependant equally on ligaments and on muscles.

If we remember that fact we shall be spared much disappointment in what I call "fancy operations" on the knee, that is, arthroplasties and reconstruction of the crucial ligaments. Several years ago I did this operation on a German boy and, as far as the ligaments went, secured a result quite as good as any shown here. The boy's spirit had been so broken however by years of disability, false diagnoses and bad treatment that he could not be persuaded, forced or cajoled into moving his knee and thereby developing his muscles. I am thus not sure that he derived any benefit from his operation. Similarly, I performed arthroplasties on two girls, one Italian and one Austrian. Technically the operation appeared successful, but no amount of physiotherapy or occupational therapy would make them move their knees. They were finally discharged. The Italian girl appeared one day walking with a barely perceptible limp, with about 35° of motion. On being questioned she blushingly confessed that she was engaged to be married,

and did not wish her fiancé to know that she was lame. Shortly after the same miracle took place in the case of the Austrian girl. Her charitable uncle got tired of supporting her in idleness and she was forced to get a job.

I think that is a point of great importance in the selection of cases suitable for such operations, that the patients should have enough iron in their souls to develop the quadriceps muscle, which is, equally with the ligaments, responsible for the stability of the knee.

Dr. Benjamin: Dr. Krida spoke of the success of 6 out of 8 cases. I happen to know of one of these cases which was done some four or five years ago. After the operation this young fellow had a job as a radio repair man, and he fell off a roof and had some injury to his knee. Subsequently he fell off a horse, and was again injured but was able to walk after that. That seems to indicate that his knee was fairly well stabilized.

Dr. Krida, *closing:* It is very important to select the proper patients for these cases. They should be under thirty years of age.

OSTEOCHONDRAL FRACTURE OF THE KNEE-JOINT*

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NEW YORK

THE case here shown is one of a number of operated cases of fractures of the articulating surface of the internal condyle of the femur, commonly entailing as a late result the formation of free osteocartilaginous bodies in the knee-joint. The condition is commonly called osteochondritis dissecans. Since there is in these cases no clear evidence of osteochondritis, and since all those cases operated upon by myself were of definitely traumatic origin, I preferred the designation osteochondral fracture, as more clearly indicating their traumatic origin, and so described it in a previous paper.[1]

A. V., aged fifty, sea captain, U. S. Marine Hospital, No. 21.

History: Three months prior to operation, he fell from a height of 5 ft., striking on the extended right lower extremity. The knee became painful and swollen but he was able to hobble about. The pain and disability became gradually worse, so that his sleep was disturbed, and he lost 15 lb. in weight. He complains of a sensation of slipping on the inner side of the joint, and he refers his pain to that point.

Examination: He walks with the aid of a stick, evidently in considerable pain. The quadriceps muscle is greatly atrophied, extension is limited at 165°, flexion is possible to 80°. There is rather slight infiltration and no effusion. There is marked tenderness over the articular surface of the internal condyle when the knee is flexed.

X-ray films show an irregularly oval fracture of the articulating surface of the internal condyle. The fracture line is represented by a narrow area of rarefaction which has been accentuated artificially in the accompanying reproduction since it may be seen well only by transmitted light in the original films.

Operation: December 14, 1927. The joint was opened by the general utility incision. The middle of the weight bearing surface of the internal condyle presented a semi-detached fracture fragment ¾ × 1 in. in size, and including about ⅛ in. of the underlying bone. This was removed; the rough edges of the remaining crater were smoothed off as well as possible with a chisel.

Result: His pain was relieved by the removal of the fragment. February 1930; range of motion complete. There is grating in the joint, and some discomfort after use, so that he has had to seek lighter employment. His symptoms have to a great extent been relieved, but those which persist must be ascribed to the location of the fracture directly in the middle of the weight bearing surface of the condyle.

[1] Krida, A. Osteochondral fracture of the knee joint. *Surg. Gynec. Obst.*, 39: 791, 1924.

* Presented before the Section of Orthopedic Surgery, New York Academy of Medicine, February, 1930.

DISCUSSION

Dr. Henry Milch: I am of the opinion that the type of osteochondral fracture due to a severe trauma is only one form of osteochondritis dissecans. Within the past three months, I have operated on a case which I consider osteochondritis dissecans in which there was a history of repeated minimal traumata. On opening the joint, found two osteochondral masses, but no concavity on the surface of either condyle from which they might have originated. The cartilage covering the articular surface of the femur gave the impression of having been moth-eaten, either as a result of the erosion caused by the joint mice, or as a result of the underlying pathological process which resulted in the formation of joint mice. I feel that this, too, is a case of osteochondritis dissecans, and that it should be included in the large group of osteochondritis dissecans which embraces as one type the osteochondral fracture which Dr. Krida has presented.

Dr. W. M. Brickner: On various occasions I have advocated the treatment by aspiration of so-called traumatic synovitis. I have no doubt some of you have also used it instead of the time-honored strapping, bandaging and massage. I employ it in the smaller joints, e.g., the finger, the wrist; but, of course, especially in the knee. I have also emphasized the fact that the fluid at the outset of these cases is always blood or bloody, in other words, that we are dealing with an acute synovitis with a hemarthrosis; and, since there is blood in the joint, we must postulate a tear of the capsule to produce it, and some injury to a structure outside the capsule, ligament, bone or cartilage. Therefore one should have a more definite picture of traumatic synovitis in mind than that of a mere sprain. The injury is often an overlooked joint fracture that may not be seen even with ordinary *x*-ray examination. Sometimes, however, we will find a crack in the bone (femur or tibia) if the pictures are taken from various angles and with particular care to get bone detail. Also productive of traumatic synovitis is the condition Dr. Krida has described, osteochondral fracture, and this, too, occurs oftener than is generally recognized. I have in mind a case I operated on some time ago, a young man with recurrent effusions in the knee-joint following an injury sustained some months previously and with an *x*-ray finding similar to that shown by Dr. Krida. He, too, had an osteochondral fracture of a condyle of the femur. When I opened the knee-joint and removed this fragment of bone and cartilage, I found another, unsuspected and very much larger fragment of partly attached condylar cartilage, a pure chondral fracture of the articular surface of the femur. There must often occur such chondral fractures or cartilage tears, in connection with traumatic synovitis. We can see roentgenographically the osteochondral fracture, since it tears off a bit of bone; the purely chondral fracture, however, is not shown in the *x*-ray film.

Fig. 1. Osteochondral fracture.

Dr. Krida, *closing:* I agree with Dr. Brickner that some of the most striking cures can be made by aspirating the knee-joint in cases of hemarthrosis. On one occasion I was called to see a big man who had been lying in bed for three days unable to obtain relief from mor-

phine; he had hurt his knee, and by having the joint aspirated of about 60 c.c. of blood, he was entirely relieved.

As to Dr. Milch's remarks about these fractures, I did not mean to imply that there if no such condition as osteochondritis dissecans; it must be very rare; in exploring knee-joints I have never seen one that I could not attribute to traumatism. I have never seen osteochondritis dissecans non-traumatic. These fractures correspond in appearance to Koenig's description of osteochondritis dissecans.

DISLOCATION BETWEEN FIFTH & SIXTH CERVICAL VERTEBRAE

DELAYED OPERATIVE REDUCTION & FUSION*

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THE case here reported is one in which a successful outcome was obtained by open reduction and fusion of a grossly unstable fifth and sixth cervical dislocation, eleven weeks after injury. The possible failure to obtain and the obvious failure to maintain reduction are outlined in the recital of events prior to the date of operation.

History: A. H., aged forty-two, laborer. Forty-eight hours previously he was struck on the back of the head by a bale of hay falling from a loft in the barn. It was stated that he was immediately disabled and that he had paralysis of the extensors of the wrists and fingers, and of the biceps humeri on both sides, together with a feeling of heaviness in both lower extremities.

I was asked to see the patient at the request of an insurance company in consultation with his attending physician at a hospital in the country. He had at this time no paralysis, but weakness of the extensors of the wrist and fingers, and a very much increased knee-jerk on the right side. He was in great pain and his neck was rigid. The *x*-ray showed a nearly complete forward dislocation of the fifth upon the sixth cervical vertebra.

Operation 1. Closed manipulation. The patient was removed to the operating room. Traction apparatus was improvised, and the patient was anesthetized with ether. Traction was applied to the head with an improvised portable Hawley apparatus, the head straps being fastened to a single foot stirrup, and counter traction by a surcingle over the iliac crests reinforced by manual traction on the lower extremities. After fairly considerable traction had been applied, there was a coarse bony click. The traction on the head was then maintained manually and a plaster of Paris dressing was applied including the chin and occiput and extending to below the nipple line.

The patient was not again seen until admission to Hospital for Ruptured and Crippled five weeks later, and details of his stay at the country hospital include an *x*-ray picture taken through the plaster collar which was reported upon as indicating that the dislocation had been reduced. Without advice the collar was removed at the end of three weeks since there was no evidence of paralysis at this time and patient allowed to sit up and move his neck about. At this time an *x*-ray was made which, much to the astonishment of the attending surgeon, showed that the dislocation has either recurred or in the initial instance not been reduced.

November 9, 1928. Hospital for Ruptured and Crippled. There was no pain, paralysis, or gross external deformity. The *x*-ray showed a remarkable degree of dislocation at the junction of the fifth and sixth cervical vertebrae. The dislocation of the fifth body was of a degree where its posterior border impinged just behind the anterior upper border of the sixth. There was also an angulation at this point in which the upper part of the spine was angulated forward on the lower. There was no evi-

* Case reported and *x*-rays shown before the Section of Orthopedic Surgery, New York Academy of Medicine, February, 1930.

FIG. 1. Condition five weeks after injury.

FIG. 2. Reduction of deformity by traction on convex stretcher frame.

FIG. 3. Recurrence of deformity in Calot jacket.

FIG. 4. Solid fusion with slight deformity six months after open operation.

dence of gross crushing or of fracture of the laminae.

He was placed on a convex stretcher frame and traction was applied to the head. After one week an *x*-ray showed a marked reduction in the deformity. After three weeks of traction a Calot plaster of Paris jacket was applied under suspension. Five days later an *x*-ray was made through the Calot jacket, which showed that the deformity had recurred to substantially the degree present on his admission to hospital. This indicated that there was no stability in the operated area, and he was prepared for open operation.

Operation 2. December 11, 1928. Operation for dislocation of the cervical spine. A long median incision, the patient's head resting in a special head rest so arranged with a traction band so that traction could be made during the operation, the band being placed around the waist of an assistant. The spinous processes and laminae were exposed beginning at the first dorsal vertebra and working upward. It was found that at the junction of the fifth and sixth cervical vertebrae, the upper ones were displaced forward and flexed to a fairly remarkable degree, the distance between the lamina of the sixth and that of the fifth being fully 1 in. and the forward displacement in this plane being about ½ in. The laminæ of the fifth, fourth and third were carefully freed and exposed. Then by combination of grasping the lamina of the fifth and levering on either side between the articular facets and by traction on the head, the fifth was gradually mobilized and drawn backwards on to the sixth. At the termination of the mobilization and reduction and upon releasing the traction on the head there was a fair tendency for the bones to remain in place. Fusion between the fifth and sixth was then done by the Hibbs method, turning up bone from both laminae and erasing the joint, splitting the spinous processes and utilizing as re-enforcing material the split spinous process of the seventh cervical vertebra. The incision was closed and the previously prepared plaster of Paris Calot collar was applied.

The postoperative *x*-ray showed that the dislocation had been reduced. The patient was discharged from the hospital at the end of six weeks, wearing a Calot collar, for which a leather Thomas collar was substituted after one month. All support was removed at the end of five months.

August 31, 1929, eight and one half months after operation, the *x*-ray shows that the fused area is solid, and that there is but slight deformity.

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EDITORIALS

FRACTURES AND MODERN HOSPITALS

THE recent visit of Prof. Lorenz Böhler of the Unfallkrankenhaus, Vienna, to demonstrate his methods in the treatment of fractures has its significance at this time. It serves to emphasize the growing importance of fracture treatment in this country and abroad and gives us the opportunity to measure his standards to ours and to determine wherein our methods can be improved. His visit comes at the opportune moment when this question is receiving serious attention from both the medical profession and the laity. His visit should be profitable to us as he has brought fresh views on the subject and has enlightened us with authoritative information on the progress that has been made in Europe.

The addresses he delivered before the American Orthopaedic Association and the American Medical Association were impressive to say the least. Many features of his method are novel in their application although not entirely original in their conception. The general use of local anesthesia, extensive use of axial bone traction and the unpadded cast all have a place in the modern treatment of fractures but their application to the methods that prevail in this country should be governed by good surgical judgment and expediency. Time, patience, and surgical skill will define their true merits but with the information now available there is every indication that the methods he advocates will find a permanent place in our treatment of fractures.

He has given ample demonstration of the outstanding principles of his treatment but to fully appreciate his success one must

visit the institution for first-hand information of his methods and organization. It becomes apparent above all else that *the hospital is the proper and logical place to undertake the efficient treatment of fractures* with the possible exception of minor bone injuries that can be treated at home. The success of Böhler's treatment at the Unfallkrankenhaus is measured in terms of surgical skill, equipment and organization, each receiving proper emphasis but with correct balance in functioning as a whole. The standards he sets for us would require the establishment of a special hospital devoted to the treatment of fractures, with full-time assistants, highly trained and efficient in the specialized care of bone injuries. The extent to which we may follow his example remains to be determined but from the knowledge gained by his demonstrations in this country there appears to be little doubt that the present hospital methods of treating fractures have not maintained the high standard of efficiency evident in other branches of surgery. In a measure this is the fruit of Böhler's message and if we are to profit by his example we must take cognizance of our omissions in so far as our hospital methods are concerned.

The modern hospital aims toward perfection of organization, specialization and standardization and without these standards of efficiency it cannot keep in step with progress. We have come to an awakening in regard to the treatment of fractures which, in a measure, has been forced upon us by the throbbing, pulsating everyday life that has grown up outside the hospital. Industry, traffic and even our methods of living are being constantly standardized, specialized and ever moving forward. The modern hospital plays a vital part in this complex social order and no effort of ours should be spared to maintain the high standards that are expected of us. Advances in medical science are not solely due to our professional initiative. They may reflect progress that originates outside our field of science. They may even bow to the demands of other influences that momentarily outstrip us in the march of progress. A demand has already come to us for improvement in our methods of treating injuries. The patient, the employee, the employer, society and industry in general call upon us for better service, better time-saving methods, more efficiency, and greater skill to minimize the devastating effect of fractures to society and industry. This demand upon the medical profession must be and will be seriously and efficiently met. There is no question of personal service involved for that has been the profession's greatest attribute. There is no question of knowledge or of skill as they are amply demonstrated in almost every other field of surgery.

We may ask ourselves why this is true of the treatment of fractures while other branches of surgery have advanced far beyond. The answer must be apparent to those who seriously seek the facts. Let us call it a state of mind into which we have allowed ourselves to lapse without fully realizing that we have ceased to progress. We have in a sense been standing still in our *methods of organization* for the treatment of bone injuries.

The remedy is within our reach and it requires but the effort to grasp it. The modern hospital is the place to initiate the organization for the treatment of these injuries as it is the immediate point of contact with the injured. Let us apply our standards of organization, specialization and standardization to the treatment of fractures as they exist in general hospitals today and determine wherein we have failed to measure up to the high ideals and humanitarian motives that guide us in our life of service to the patient.

To a small group of surgeons specialization in the modern treatment of fractures is an accomplished fact and the character of their work is worthy of the highest commendation. *To many more* it is still an ambition while the *rank and file* who have not as yet given serious thought to

the subject still adhere to the belief that the clinical surgeon is fully capable to treat all fractures as part of the general practice of surgery. Faith in this belief is rapidly waning as the extent and character of the advances in this field of surgery are more generally known. Doubt casts a glance at this belief of surgical efficiency and further speculation might even question the proof of this claim. Compensation bureaus and industry have already offered facts to the contrary and measures have been suggested to meet the situation. While the treatment of fractures has failed to assume the commanding position it deserves there has developed a serious situation that vitally affects the interest of the profession due to the surgeons lack of understanding of its scope and magnitude. Commercial agencies are already knocking at the doors of our hospitals for the privilege of treating fractures with special patented devices and even furnish the necessary service for their application for a fixed minimum charge to the patient.

Our efforts must be devoted to furnishing the patient with the most efficient service and to restore him to health in the shortest possible time. The present organization for the treatment of fractures leaves much to be desired and very radical changes may be required before our surgical initiative has been re-established. Let us seek and welcome more surgeons with messages like Professor Böhler's and let us stir ourselves to greater efforts to maintain the high standard of bone surgery that has been associated with the names of countless number of American surgeons whose achievements are an inspiration and a source of just pride.

Professor Böhler returns home with our sincere expression of appreciation and hearty commendation for the excellent work he has performed in his special field of surgery. He has been a perfect guest. He has done the things we wished him to do and he has told us the things we wished to know. He has been untiring in his efforts and gracious in his manner and we sincerely hope and trust that his first visit to this country has been a happy one that will furnish him pleasant memories and lasting friendships but that it will be the forerunner of many others of a similar nature.

William G. Doran, M.D.

MEDICAL TEACHING

Walter Alvarez has just had published a most interesting and instructive book on "Nervous Indigestion."[1] We take the liberty of quoting from parts of his Preface. Some months ago John Osborn Polak wrote an editorial on The Clinical Teacher.[2] It caused widespread comment. We received scores of letters, most of them approving Dr. Polak's stand, which made us think this subject is a live one in the minds of many men.

Thus, while reading the preface to Alvarez' book we could not refrain from marking parts of it. Without further editorial comment we will quote the paragraphs checked by us. The reader may draw his own conclusions.

As time goes on it seems probable that most teachers of medicine will step directly from the laboratory into the professional chair, and as a result there will be fewer and fewer of them with any knowledge of medicine as it is practiced in a downtown office, with patients from the middle and upper classes. This will be unfortunate because most of the students in

[1] Dr. Alvarez' book, "Nervous Indigestion," is reviewed under Book Reviews in this number of The American Journal of Surgery, p. 578.

[2] Am. J. Surg., 7: 723, 1929.

our universities are going out into private practice, and I think they should be prepared for the fact that they will meet with human beings and problems different from those encountered in clinic and ward . . . It seems to me as time goes on more provision will have to be made for acquainting medical students with the problems that they are going to meet in practice . . . I doubt if teachers of medicine realize how seldom it occurs to them to spend an hour in the amphitheater talking about "functional" disease . . . If these conditions continue I fear that every year more and more students will graduate from even our best universities well equipped with information about the rarer diseases, which they will seldom encounter, and woefully unprepared to deal with the functional troubles which they will see several times a day. Furthermore, they will have learned much of the science of medicine but little if anything of the art.

T. S. W.

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SIR CHARLES BELL

[1774-1842]

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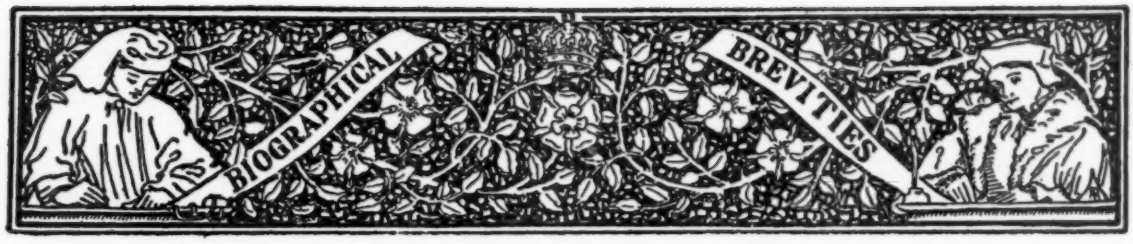

"BELL'S PALSY"

AN artist of ability, an anatomist of distinction, a surgeon of rare skill, and a man popular with his professional colleagues and society, sums up the character of Charles Bell.

He was the son of a Scotch Episcopal clergyman. John Bell, a surgeon of reputation, was his brother.

Charles journeyed to London in 1804. He opened classes for the teaching of anatomy at his own home and later at Great Windmill Street. In addition he gave courses of lectures to artists. The result of these lectures became embodied in his "Anatomy of Expression" (1806).

Because he devoted the major part of his time to private investigation he had little time to acquire a private practice. In the end he accepted the chair of surgery at Edinburgh in 1836. What the laymen of London missed was more than supplemented by Bell's achievements of a scientific nature.

Bell illustrated his "System of Dissections" in 1798. He did "Engravings of the Brain and Nervous System" in 1802. His Bridgewater treatise on the hand, done in 1833, is remarkable because of his exquisite sketches. In 1811, he published "A New Idea of the Anatomy of the Brain and Nervous System." In this work one finds an epoch-making sentence.

> On laying bare the roots of the spinal nerves, I found that I could cut across the posterior fasciculus of nerves which took its origin from the posterior portion of the spinal marrow without convulsing the muscles of the back, but that, on touching the anterior fasciculus with the point of the knife, the muscles of the back were immediately convulsed.

So we have the first recorded experimental reference to the function of the spinal cord in literature.

Although Bell made many discoveries he had a dislike for vivisection. Therefore, he failed to intrepret many of his findings. For instance, it was left to Magendie in 1822 who used a litter of pups to find that the anterior roots are motor and the posterior roots sensory.

In 1829 he demonstrated that the fifth cranial nerve is sensory-motor. At this time he discovered "Bell's nerve." We refer to "Bell's palsy" from his discovery that a lesion of the portio dura of the seventh nerve causes facial paralysis. He incorporated all these discoveries in a book on the nervous system in 1830. This work contains early reported accounts of pseudo-hypertrophic paralysis and "Thomsen's disease."

Bell was in his day known as a dandy. By nature genial and sociable he was much lionized by London society. In 1829 he was knighted for his physiological discoveries. We read that as a surgeon he attended the wounded after the battles of Corunna and Waterloo, and left many interesting sketches of the things he saw.

All in all his was a full and interesting life. Born in 1774 he died at the age of sixty-eight years in 1842.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

SOUVENIRS OF MEDICAL REGISTRATION IN FRANCE*

THEODORE C. MERRILL, M.D.

PARIS, FRANCE

THIS account presents experiences which depart but very little from those which must be encountered at the present time by any foreign candidate for official medical practice in France. The viewpoint represented is, naturally, mainly an Anglo-Saxon or American one and the coloring is possibly not the same as would be the case were the reporter a Latin, Oriental or Slav. However, the procedures themselves are alike for aspirants of any race or nationality.

FRENCH MEDICAL DIPLOMAS

All courses of study offered in the French universities of the capital and provinces are practically parallel and uniform, being prepared and formulated by the central Ministry of Public Instruction. Differences are due principally to variations in the extent and variety of clinical material. Mainly for this reason, the Faculty at Paris is considered the most important of the French medical teaching groups. However, the Faculties at Strasbourg, Lyons, Marseilles, Montpellier, Bordeaux and Lille are also of high excellence. Because of the centralization and uniformity prevailing in the French educational system, diplomas granted by the medical faculties of all the French universities enjoy equality of official recognition and confer equal rights upon those possessing them.

The French faculties award diplomas of two kinds. First, they may grant the *diplôme universitaire.* This diploma is awarded to persons desiring a formal French certificate or diploma, referring to general medical study (as contrasted with limited and special post-graduate work) and serviceable at home or abroad as evidence of study done in France. This diploma does not entitle its possessor to practice medicine in France or the French colonies. It is obtainable by physicians who are graduates of acceptable medical schools, provided the candidates pursue the regular undergraduate medical courses of at least the last two years, perform the accompanying work in hospitals and laboratories, pass the regular didactic and clinical examinations and present and defend the thesis which is invariably required. Aspirants for the "diplôme universitaire" are not obliged to obtain the French academic baccalaureate degree or the so-called "P.C.N.," a certificate showing one year's work in physics, chemistry and the natural sciences.

For the right to practice medicine or surgery in France, the *diplôme d'état,* or official state diploma, is absolutely

* Submitted for publication April 10, 1930.

necessary. It is granted with the legal proviso that candidates for it must possess the French baccalaureate degree and the P.C.N. certificate just mentioned, must complete at least the two final years of the regular medical curriculum, must pass the examinations on the work of these two years and the final clinical tests in medicine, surgery and obstetrics, must fulfill the hospital work or stages accompanying the two years' medical study and must present and defend an acceptable thesis.

Requirements for the *diplôme d'état* are thus exacting. They allow no credit for previous college or univeristy work abroad, except that they omit, for physicians of good standing in their own home countries, the first three years of the regular medical curriculum and do not actually insist on the year's work for the P.C.N. certificate. For once this diploma has been obtained, the shortest possible period of study ranges from four and one-half to five or more years, according to the candidate's facility in doing the baccalaureate work, passing the medical tests and completing the thesis. Such was the program followed very literally by the writer when engaged in solving the problem of entering upon medical practice in France.

THE BACCALAUREATE

This degree roughly corresponds in scope to the work of the first three years in American colleges for the A.B., PH.B. or B.S. degrees or their equivalents. It may be obtained either before or after completion of the medical work. The writer attacked it before.

In a naive hope that fairly fresh remembrance of former college work done in the United States might be sufficient, the examination for the first part of the baccalaureate was rather rashly attempted in the autumn, after a rapid review of only some three weeks. The natural, and instructive, result was failure, to an extent which showed, however, that success might not be impossible with adequate preparation.

The studies for the baccalaureate may refer either to sciences, languages and literature or to sciences and mathematics. The writer chose the former group, or *Latin-Langues*, the second group requiring mathematics extending well beyond mathematics given during the freshman and sophomore years in American colleges. The necessary preparation was directed by a competent private French instructor. The writer was thus classed as an *élève libre*, or free student, as contrasted with a student attending regularly outlined courses in a French secondary school, or lycée. Instruction may also be sought at various schools which advertise more or less widely. These schools, however, of commercial type, are graphically known as *boîtes à bachaut* or mere *baccalaureate boxes*, and I believe that the study which they supply is too summary and rapid for foreigners and not solid enough to give the foreign student much assurance of success in passing the official examinations. The *boîtes à bachaut* are bad enough, even for native French students.

The writer's instructor gave a weekly lesson of two hours' time. It dealt with a variety of subjects. The weekly lessons were continued without a break during the two-year period devoted to the baccalaureate, obtained after passing the regular examinations at the Sorbonne.

The first-year subjects were French composition, the drill referring to French history and literature; general history of the eighteenth century, Latin, geography, and mathematics through solid geometry. For the second year, the studies were in philosophy, the authors selected being Descartes, William James, Claude Bernard, Ribot, Janet, Bernstein and others; general history of the nineteenth century, German, English, physics, chemistry, mathematics, botany, zoology, elementary human anatomy and physiology and a continuation of French composition, devoted mainly to philosophical and psycho-

logical material. In the *Latin-Langues* course mentioned, two foreign languages are required, those of the writer being

Fig. 1.

English and German, the native language supposedly consisting of French.

Intensive review and study are necessary for confronting the examinations for the baccalaureate, especially if the candidate is not freshly familiar with his material. Such review is important, even if the material is more or less familiar, because it presents new viewpoints, promotes good knowledge of French and brings out peculiarities inherent in French examinations and the presentation of the subject-matter. French standards are strict. However superfluous some methods may seem, they must be complied with. Conventions must be respected. In academic tests, original theories or opinions are neither desired nor accepted. Discussions should avoid reference to religious, military, political or other points of acutely present interest or controversy. The examinations are designed to show the candidate's accomplishments in the prescribed educational program. The latter, by the way, is so uniform throughout France that it is often said, and without too much exaggeration, that instructors know, at any given moment, precisely what page in any subject is being studied by all the students of France of the corresponding grade.

During his training-period, the writer referred, in a certain composition, to Poincaré, Marshal Foch and the French President. The instructor raised both hands in shocked protest. "Never," said he, as he vigorously scratched these names, "never discuss, for such an examination, anybody who hasn't been dead for seventy-five years at least!" Introduction of personal or controversial views into French examinations is sometimes very disastrous.

BACCALAUREATE EXAMINATIONS

At the close of the first study-year, a *convocation* or summons, was received by mail, due application for registry in the yearly examination having been filed two or three weeks before, on officially stamped paper, with presentation of birth certificate. The writer's convocation was worded as follows:

"Université de Paris, Faculté de Lettres; Baccalauréat Latin-Langues, First Part. No candidate will be admitted without his receipt for the examination charges. Written examination 7 A.M., July 12. Oral examination 2.30 P.M., July 18. Only candidates succeeding in the written tests are admitted to the oral examinations."

On the morning of July 12, then, the author joined a throng of young men and women, aged, in general, from sixteen to twenty, who were surging through the corridors of the Sorbonne in search of the amphitheaters to which they had been assigned. The young folks' faces were grave, and so were those of the parents and friends who accompanied them, for failure here means the loss of a whole year, or at least painful drudgery through the summer for supplementary examinations held in October.

Doors were not opened until about

eight o'clock. The students then filed into rooms full of separate desks and tables. Each student deposited his *livret scolaire*, written tests occupied two days. No marks were immediately furnished, the students remaining ignorant of success or

Fig. 2.

or record book, signed by the school or instructor who had prepared him, submitted his fee receipt and received a number. The questions were read aloud. Should a student fail to understand, he may ask to see the printed slips bearing the questions. These slips are in the hands of the examining officer. That it is well to be sure of the questions is proved by two curious errors recently committed. On these occasions, subjects allotted for discussion were the following: "Victor Hugo sera lu, mais en anthologie [Victor Hugo will be read, but in anthologies]" and "Hygiène de la mémoire [hygiene of the memory]." Two respective students understood the examiner to say, "Victor Hugo se rallumait en entrant au logis [Victor Hugo grew light-hearted on coming home]" and "Hygiéne de la baignoire [hygiene of the bath-tub]." The papers of these two unfortunates were marked zero in the subject of literature.

A choice between several groups of questions is usually offered. Writing-paper is the only material supplied by the authorties, and waste of it is not welcomed. The failure until the moment of the oral tests. With these, the anxiety of the assembled students seemed even greater than before. Not until the hour set for the orals did monitors appear, posting lists of those who had passed the written examinations and who might, therefore, be admitted to the oral ones. As the students crowded forward to read these lists, painful indeed was it to remark the disappointment of those who had failed and to read the downcast faces of their parents and friends as they and their unlucky candidates turned sadly away from the Sorbonne battleground, either to face still further preparation or to give up the struggle for a technical career. Failure is, of course, still possible for students admitted to the orals.

The examiners constitute a species of jury and are known as a jury. Each member presides over his own special subject and the students appear before him one by one. At the close of the test, the grades obtained by each student were read aloud to all and the students were then dismissed in a few gracious and kindly

words uttered by the President. Each student recovered his *livret scolaire* before leaving the room, and the first part of the examination for the *bachaut* had become a thing of the past.

Fig. 3.

The second part was completed in similar fashion. At the oral test, the professor of history was intrigued by the appearance of an evidently older (and foreign) student among the young stock, but exchanged cards and courteously invited the stranger to make his personal acquaintance and wander now and then through sections of Old Paris, dear to the historian's heart. This was one very charming and lovely fruit of the baccalaureate inquisition.

A provisional diploma was received some two weeks after passing the oral tests of the second part of the *bachaut*. The final official document was not received until a year or so later. Delivery was slow because of scarcity of university funds and personnel, in evidence ever since the difficulties of the war. The provisional diploma served for presentation when applying for medical enrollment.

MEDICAL ENROLLMENT

Here I obtained first-hand information concerning the interesting and important question of influence, "pull," or *piston*, as the French call it, in its relations to the abbreviation of requirements. I may as well say at once, bluntly and frankly, that it is better to depend solely upon oneself and to disregard all advice counselling the application of *piston*. Formal regulations, of which printed copies (such as the *Livret de l'Etudiant*, published by the Presses Universitaires) are available at the medical bookstores, clearly indicate the necessary procedure. Pull may work well, or may have worked well, in certain cases but it did not with me and it is not generally effective, its relation to my own case being illuminating enough to justify a tale of my experience.

When I was about to enroll, certain friends advised me to utilize the services of friends of friends who were in official employ. It was not easy to refuse these well-meant offers and letters of recommendation were accordingly placed in the hands of a member of the Ministry of Public Instruction. This official directed me to file my papers with his attorney, who showed me how to fill out an enrollment form and then lapsed into silence. The silence continued darkly, even after October arrived and the opening of the

medical courses became imminent. My sponsor then proved to be absent on his vacation and I had to act immediately. I hastened to the attorney, paid him a respectable fee for very little assistance and was obliged to withdraw my papers from him and present them myself to the secretariat of the Faculté de Médecine.

My letters and other papers were transmitted thence to the appropriate Faculty committee, but were returned to me without the slightest sign that they had even been opened. They were ignored, in fact, because of the attitude of the French Syndicat des Médecins, or physicians' union, which actively safeguards French medical practice and practitioners, as it should do in order to prevent overwhelming of the French profession. The delay incurred by my attempt at "pistonning" retarded my hospital registration and I had to make up the lost time during the Christmas vacation. In fact, I was just able to complete my first hospital stage on time and narrowly missed losing two months. It would have been far preferable to act directly.

After I had enrolled for the medical work, the required P.C.N. certificate proved to be obtainable *à titre onéreux*, which means that it would be granted me on payment of the customary fee, with absolution from the extra year of work represented by it. This concession was, naturally, welcome. The principle of common sense was here delightfully given full play. With the baccalaureate diploma and the P.C.N. certificate, medical enrollment could proceed without difficulty, save with respect to the afore-mentioned tardiness in arranging the first hospital stage.

Each medical student is supplied, on enrolling, with a *livret universitaire*, a counterpart of the undergraduate *livret scolaire*. It contains his photograph, registration number and blank pages for recording his examination grades. The *livret* must always be presented to examiners, who enter within it the grades which they grant. When completed, this little book constitutes a record of compliance with regulations, of the marks obtained and of the grade accorded the student's thesis. It is finally retained by the student, whether he succeed or fail.

THE MEDICAL PROGRAM

The foreign candidate for medical practice in France is now confronting his last two years of the regular five years of the French medical system, his laboratory and hospital requirements, his coming didactic and clinical examinations, and his thesis.

Didactic Work: No attendance on lectures is required. The student is free to do as he likes in this respect, just as he is free to study or not. However, attendance at the faculty lectures is of supreme value and importance, especially for foreign students. Faithful attendance alone permits a knowledge of technical terms and of French methods of presenting written material, for examinations and other purposes. It is invaluable for such work as prescription-writing in French. Again, it is practically indispensable in preparing for the more difficult examinations, such as the tests in therapeutics, which are a special bête noire even to the French students themselves. For the examinations, lecture-attendance is highly useful, from the viewpoint of matter as well as that of manner. Foreign students who fail to attend all lectures bearing on the curriculum seriously handicap themselves. The regular French students generally permit themselves no such handicap, as proved by the crowded amphitheaters.

Nor is immediate utility all. It is not too much to say that many of the lectures given gratis at the University of Paris, and at other French universities, have no superiors in the world for brilliance, precision and charm. Seldom has the writer listened to expositions so clear, forceful and beautiful, and so full of learning, wit, humor and pure delight. Among those who eagerly seek places at these lectures are

not only undergraduate medical students, but physicians, surgeons and technicians of every race and language. Places are decidedly at a premium in some of the lectures. At those on therapeutics, given in the large amphitheater of the Paris faculty, every place was always filled and "standing room only" awaited late arrivals. In my own case, desiring a seat where I could hear clearly and have a good view of blackboard notes, it proved necessary to appear fifteen minutes before the opening of the lecture. The few inevitable departures from this practice were always deeply regretted.

Laboratory Work: The required laboratory work, much the same from year to year, refers to pharmacology, medical chemistry, pathology, study of sections, legal medicine and toxicology, and hygiene. An optional course, of decided value, is given in prescription-writing and is of special utility for those intending to practice medicine in France or the French colonies. The work in the subjects just mentioned comes during the last two years of the medical curriculum, other laboratory work being also included in the first three years of the regular medical program.

Hospital Stages: Regular daily hospital attendance is insisted on from the beginning of the French medical training. In my case, the required work progressed chronologically as follows, each period or stage, being of two months, except in the subject of obstetrics, in which three months constituted the period: infectious diseases, at the Claude Bernard hospital, used for isolation; dermatology, at the Hôpital Saint Louis; neurology, at the Salpêtrière; obstetrics, at the Hôpital Boucicaut; psychiatry, at the huge asylum of Sainte Anne; ear, nose and throat, at the Hôpital Lariboisière; ophthalmology, at the Hôtel Dieu and urology, at the Hòpital Necker. The professors in charge were, respectively, Teissier, Jeanselme, Guillain, Le Lorier, Claude, Sèbileau, Terrien and Legueu.

In the hospital stages, attendance is usually checked by roll-call and record. At the Claude Bernard hospital, the students had to sign one roster at the beginning and another at the end, of each morning's work. Absences count seriously against the student if they are detected. Each stage must be marked and certified by the professor or *chef de clinique* in charge before the student can be admitted to the next stage in his program. Quizzes are held more or less regularly. Lectures and various demonstrations are given during the stages. The student should familiarize himself, by outside reading and study, with the pathology and therapeutics applicable to the given stage which he may be pursuing.

Final Examinations: These are of two kinds. First, there are those held at the close of the required subjects. These are generally oral, but the examination in therapeutics usually includes a preliminary written test. If this is passed with a grade of 70 per cent or over, the candidate is excused from the oral examination. The less well prepared students are thus confronted with two examinations in this subject.

The oral tests are interesting and, for some of the students, fearsome. However, they are perfectly fair and students who cannot satisfactorily answer the classical questions put to them may be safely considered incompetent. The jury system prevails. The student usually finds three professors in charge of the examination. Interrogated according to lists held by the examiners, each student appears before one of the three. There can be no question whatever of favoritism, for the students are examined in alphabetical order and assigned to the different juries only on that basis. Indeed, the student and his examiner will never have met before, unless some chance has brought the student into contact with the professor during a preceding hospital stage, or unless the student may have seen the professor from afar during some lecture given by the professor. In practice, examiners are nearly

always without the slightest acquaintance with the students who appear before them.

Arrived before the examiner, the student replies to one or two perfectly fair questions. If the reply shows sufficient proficiency in the given subject, the examiner writes in the student's "livret" the grade obtained and the student is replaced by another. The grades are *très bien* (roughly 90 to 100 per cent), *bien* (80 to 90 per cent), *assez bien* (70 to 80 per cent), and *passable* (60 to 70 per cent). Should the student fail to satisfy the examiner, he is referred to a second one and may even have a third try. However, reference to the second examiner means still more searching questions and augurs ill for the student.

All examinations are public. Any who desire may remain until the last student has been examined. Students and others present then leave the room to the jury, who consult, more especially on students of doubtful attainments. After the jury have completed their deliberations and departed, the *garçon*, or aide, distributes the students' record books, or *livrets*, and often reads aloud the marks received. Low marks elicit sympathy and complaint, while high marks are greeted with cheers and felicitations.

A second variety of examinations is constituted by the final clinical tests in medicine, surgery and obstetrics. They may be attempted whenever the student is ready for them, and in any order preferred, after due application. A few days after the first test has been applied for, the student's name appears listed on the Faculty bulletin board. Each student makes one of a small group assigned to some hospital with which no student of the group is supposed to be familiar.

The clinical tests are not always easy and are a source of more or less anxiety, though well-prepared students have little to fear. On arriving at the hospital, the group is taken in charge by the professor or other responsible chief. To each student is assigned a case, taken haphazard from the wards. About fifteen minutes are allowed for examination, diagnosis and outline of the indicated treatment. The student makes his record in writing, seals it and hands it to the medical officer in charge. On the next day, he undergoes a similar test at another hospital, his second case naturally differing from his first. Each examination, medical, surgical or obstetrical, thus includes two cases. The two clinical examinations in each subject are given rather early in the week and on the following Saturday the student repairs to the medical school, where the physicians in charge of his group assemble to form the usual jury. The sealed envelopes are then opened and the student discusses his case and his records, the interrogator being familiar with the case. If the discussion and record are satisfactory, the mark received is inscribed in the *livret* and the student is dismissed with kindly words. Each student thus appears before at least two professors and may be assigned to a third if his status seem doubtful. The professors are frank and outspoken, though by no means finicky or unjust. "Eh bien," remarks one, "don't look at me, look at your paper. You can't read the answer in my face." "Why do you prescribe that?" asks another. As the student stammers that his choice was made on the basis of home practice, the professor rejoins, "Remember that this is Paris, not Uruguay!" "Why," exclaims a third, "you don't seem to know much about this. Just describe the operation which you advise."

Well-prepared students may finish all three clinical tests in about six weeks as a minimal period, some allowance being necessary on account of the examinations of other students and intervening holiday or vacation periods. Most students devote more or less time to special preparation for the clinical tests, which may thus extend over several months. Some professors are dreaded, others are preferred. However, the student must take his

chances and has no choice in the selection of the jury before whom he must appear.

Very naturally, all examinations are given in French. On one occasion I remember, a foreign student, not very familiar with French, asked for an interpreter. His request not only embarrassed and prejudiced the jury, but elicited loud protests from the students themselves. "No special privilege!" is a student's slogan universally insisted upon in France, except in such wise as may permit one student to aid another. Dishonesty at examinations is severely penalized, but considered lightly by students of mediocre ability or industry. In accordance with the strong esprit de corps existing among students, able ones seldom refuse to assist their less fortunate comrades, although good students do not respect shirking, bluffing, incompetence or stupidity.

Thesis: This is invariably required of all candidates for French medical degrees. It is based upon work assigned or accepted by some professor selected by the student. This professor constitutes the thesis President, or President of the jury before whom the thesis will be sustained. The thesis may represent prolonged and valuable original work, or it may be prepared in a more or less rapid, summary and perfunctory fashion. However, it must be accepted by the President, who may, if he likes, assign a task requiring a year or more for its completion. Prolonged study is the rule with students who are interested in some special subject and who are in a position to do very thorough work, without being in any haste to begin practice. Such is the case with French internes, who have ample opportunity for extensive research during their usual five-year period, and who are not legally permitted to undertake private practice during their internship. Foreign candidates can usually prepare acceptable theses after some three months or more of work, especially if they are already in possession of data previously accumulated and acceptable to the thesis President.

Only after the three clinical examinations, already described, have been completed may the candidate proceed to the formalities attending the preparation of his thesis, although work on the latter may have been already done, wholly or in part. The professor selected having consented to act as President, and the thesis having been prepared in manuscript, the procedures indicated below may be complied with.

The manuscript and conclusions, signed by the thesis President, are filed with the secrétariat of the Faculty. The manuscript is then returned within forty-eight hours, bearing official permission to print. The candidate must bear the expense of printing and may select any printer preferred. The format of the thesis must be octavo. Theses for the *diplôme d'Etat* must be so designated on the cover. The *universitaire* theses must bear the expression, *Thèse universitaire, mention médecine.* Close attention should be paid to the latter points, otherwise embarrassment may occur. The printers are supposedly familiar with the procedure required for the printing of theses, yet they may make absurd and unpardonable errors. The candidate may consult, at any time, the official regulations, which are always left open to his use in the hall of the secrétariat. He should not fail to insist that they be followed precisely. In my own case, the printer, after assuring me that he would faithfully follow every detail, made the crass mistake of labeling my thesis *Universitaire.* Had I not been carefully following every step in the printing, copies wrongly labeled would have been filed with the Faculty librarian for general distribution and I might have had much difficulty in establishing my status as a candidate for official practice. Again, checking of the printing is especially necessary when the material contains technical formulae. During the printing, nothing should be left to chance and proofs should be carefully read and corrected.

When sure that the proof sheets are acceptable, and before the printing of the

desired number of theses, the candidate must file with the secrétariat a proof-sheet of the first page, which must bear the candidate's surname and given name, date and place of birth, the name and titles of the thesis President and a list of the professors who have presided over the student's hospital and other work. When these data have been filed, the thesis may at last be printed. At least one hundred and five copies are required, for this number must be deposited with the librarian of the Faculty for record and for distribution of a certain number of copies to different libraries or universities habitually receiving the copies from the university where the thesis is prepared. After filing the library copies, the candidate may pay the university fee required in connection with the thesis. Within two weeks after making this payment, the candidate will be assigned a date for defending his thesis, the last step preceding issuance of the coveted diploma.

Defense of the thesis is something of a ceremony. The candidate is interrogated by any or all of three jury members, of whom the thesis-President naturally occupies first place. The members wear their scholastic robes and caps. The occasion is open to the public, but there are usually no attendants except the candidates and one or two of their special friends. Students sometimes attend for the purpose of familiarizing themselves with the procedure. The ceremony is usually merely formal, for no difficulty should occur when the candidate has reached this point of his progress. However, I have heard sharp rebuke administered to a candidate who had not taken the pains to verify the formulas which he employed. The jury members promptly noticed the fact that the formulas, as expressed, were meaningless and roundly criticised the thesis for these imperfections, assigning the lowest passing-mark to this work.

When the interrogation is completed, the jury members shake hands with the successful student, express a few congratulations and pass on to the next examinee. Not more than three or four are usually interrogated at the same séance. When the ceremony is concluded, the inevitable garçon informs the new doctors of the marks they have received and pockets the small tips bestowed by the survivors of this final ordeal. The diploma may now be claimed at the secrétariat of the Faculty. When the latter has been duly registered at the préfecture of police and at the police station, or commissariat, in the quarter where the new physician is to reside, the latter has become an actual member of the French corps médical and may begin his practice.

Many cherished details scarcely come within the scope of this paper, which must omit various charming or vexatious incidents which imparted unforgettable savor to the memories of the assault upon the walls of the Sorbonne and the siege of the inner and higher citadel constituted by the Ecole de Médecine. Another occasion must be reserved for amusing tales of student life, charming relations with members of the Faculty, battles with garçons and functionaries and thrills experienced by sharing in an immense and victorious strike of the students, organized to maintain medieval law and traditions of the University of Paris.

The entire experience was well worth while and leaves most delightful impressions and memories of the Paris Faculty of Medicine and the student-body of the University of Paris, seasoned exemplars and ardent initiates of traditions and principles which have marked, brilliantly and indelibly, the science and art of medicine.

BOOK REVIEWS

NERVOUS INDIGESTION. By Walter C. Alvarez, M.D., N. Y., Paul B. Hoeber, Inc., 1930.

The debate is as old as the hills: Do book-reviewers read the books they review? I cannot answer for my neighbor but in this instance let me say I have read Alvarez' book from cover to cover. In fact, parts of it I have reread two or three times. In order that we start with the proper understanding necessary for faith and trust between writer and reader let me confess I am not a gastroenterologist. For this reason I think the opinions offered may be the more valuable. Over 90 per cent of the specialists in gastroenterology will read "Nervous Indigestion" for the simple reason they always read everything Alvarez writes. So this is not written for them. It is the man doing internal medicine, gynecology, general surgery, neurology and the other divisions of medicine I would reach.

To me this is a valuable book. It is valuable because Alvarez has chosen a subject which has been neglected. He has covered the field in admirable fashion. It is valuable because the author has not had an eye on a score of text-books and written in the dry, didactic manner so common in most scientific authors. As far as style goes Alvarez has a style all his own. His writing flows; it is always entertaining while instructive. We read his book at two sittings.

The first chapter considers Ways in Which Emotion Can Affect the Digestive Tract. It opens with two queries: "What does a physician mean when he says that a patient has nervous indigestion? Just how does the tired brain produce distress in the abdomen?" We will quote a paragraph or two:

> The most striking evidence of a psychic increase in tone activity was seen in a jolly bon-vivant whose anal sphincters had been destroyed by a series of operations for fistulas. As a result, many of the rush waves down the bowel, instead of stopping as they normally do at the ileocecal sphincter or somewhere in the colon, ran on and produced a bowel movement. The interesting point is that in this man the sight and smell and even the thought of food produced rushes.

Chapter II deals with Types of Indigestion. Alvarez says, "Nervous indigestion is a convenient term with which to designate all those gastrointestinal disturbances for which no organic cause can be found." The author goes deeply into this subject and then considers Gall Bladder Disease, Peptic Ulcer, Appendicitis [the subject of chronic appendicitis is worthy of more than passing note], Cancer of the Stomach, Cancer of the Colon, Diverticulitis of the Colon, and Intestinal Parasites. Under General Diseases we read, "There are a number of patients who think they have indigestion because they want to belch or because they feel as if gas were pressing up under the heart. Not infrequently these symptoms are due not to indigestion but to *high blood pressure* or to the distress of a *failing heart*. Obviously such patients cannot be helped by dieting but must be made to rest . . . " The pages on Fatigue Neurosis, Tempermental Indigestion, Anxiety Neurosis and Nervous Vomiting I especially recommend to you. Under enteroptosis we mentally applauded when we read, "I think I would as willingly ascribe symptoms to a large navel, to a hooked nose, or to flaring ears, as to a mobile cecum or to a redundant sigmoid flexure. The bowel is a muscular tube and I doubt very much if its position in the abdomen has much influence on the rate at which its contents are passed onward." . . . "This [mucous colitis] is another condition which accounts for much unnecessary opening of the abdomen." Alvarez reaches a high water-mark of writing when dealing with Constipation. He shows the dangers of the usual enemas. He says the patient should be taught to use nothing but a physiologic salt solution for washing out the lower bowel. Regarding diets he writes, "It is fashionable nowadays to treat constipation by the addition to the diet of bran, rough fruits, vegetables and other avowedly indigestible substances, but as might be expected, these materials often cause trouble. Those who have the digestion of an ostrich can stand filling the bowel with refuse . . . The use of hydrocarbon oil is also inadvisable in many cases . . . "

Hints in Regard to the Talking of a History comprises Chapter III. The following chapter is The Handling of the Nervous Patient. Alvarez says:

> I have always felt sympathy for anyone with "nervous indigestion" . . . I feel sorry for those who while seeking help and guidance, fall into hands of men who wittingly or unwittingly rob them, maltreat them, and make them worse . . . Instead of being given sedatives and a better diet they are rushed to the hospital, there to part with an innocent appendix, harmless tonsils, or doubtful teeth . . . Sometimes it works but too often it doesn't.

Every physician may read with profit the chapters under Behavior of Nervous Patients. Throughout this remarkable book are hints on the many methods of handling all types of patients. Every young physician just opening an office will find much of intrinsic value, matter for reflection that might well be taken to heart and accepted rather than learning these bits of wisdom offered by the long, hard route of experience. For instance the line, "I have often thought that if a physician only knew just what to say and what not to say to each patient his success would be phenomenal" and the discussion following this observation is invaluable.

The next chapter deals with The Treatment of Nervous Indigestion. The subheadings of this chapter are: Symptoms are Real: What to Say: Methods of Handling Hysterical Patients: Avoiding Deception of the Patient: Psychotherapy: Rest: The Rest Cure: Sleep: Physiotherapy, Exercise and Massage: Eye Examination: The Importance of Teeth: Diet ("Nowadays one of the simplest ways of helping dyspeptic persons is to take away their bran mush and bran muffins.").

Chapter VI considers Some Practical Points About the Physiology and Innervation of the Digestive Tract. This may sound dull and make the reader think back to sleepy days in the laboratory or anatomic quiz-room. Far from it. How I wish Alvarez could have written my textbook on anatomy and taught me physiology!

The book ends with a chapter devoted to Suggestions For Further Reading.

The Index is complete.

Perhaps I have waxed too enthusiastic and written and quoted at too great lengths, but it is rare these days to enjoy reading a scientific book although that book may be a necessity on our library shelves; but when one finds a valuable book so well written and so full of wise philosophy and so human in its scope as this one by Alvarez one cannot help crying its virtues to the world. And this we hope we have done.

MEDICAL HISTORY OF MICHIGAN. Vol. I. Compiled and Edited by a Committee, C. B. Burr, M.D., Chairman, and Published under the Auspices of the Michigan State Medical Society. Minneapolis, Bruce Pub. Co., 1930.

The increase of the interest in the medical history of this country, and the desire to render permanent the records of its beginnings before it is too late, are well shown in this large and handsome volume published under the auspices of the Medical Society of the State of Michigan. The work has been thoroughly and excellently carried out by the committee appointed for that purpose and it is evident that the knowledge and ability of its chairman, Dr. C. B. Burr, have had much to do with the success of the project. Although Michigan was a wild and sparsely settled country until well towards the end of the first half of the nineteenth century the communities which have since become its great cities were largely composed of men of ability and force of character and among them were a number of medical men who not only practiced their profession but also took an active part in public affairs. To these men we owe the foundation of medical schools like that of the University of Medicine and the high standing which the profession attained in Michigan at an early date and has successfully maintained to the present day.

Before the settlement of what is now the State of Michigan there is much of interest in the medical affairs of the pioneer French explorers and the Indians. In Chapters II and III of this book Dr. Burr reviews the available records. Considerable is known of Indian medicine partly from the pioneer settlers and partly from subsequent scientific investigations. It is curious to find that the Michigan Indians practiced trephining just as similar operations were performed by the aborigines of Peru, in the South Sea Islands, and elsewhere throughout the world. The French explorers were in many instances accompanied by surgeons and Dr. Burr gives interesting details of their experiences. One of them Liotot murdered the Sieur Moranget, La Salle's nephew, with an axe, in the course of La Salle's expedition to Louisiana in 1687. Later Liotot was himself murdered in a dispute with another member of the expedition. In the next chapter Dr. Burr tells of some physicians who practiced, chiefly as surgeons, in the Fort at Detroit. These were French until 1760 when the English took over the fort and settlement. When the United States occupied Detroit in 1796 the surgeons at the post were for many years the chief physicians in the town.

A United States army surgeon, William

Beaumont, won immortal fame at Mackinac in 1822, when he was acute enough to grasp the opportunity afforded by a gastric fistula following a gunshot wound in the side of a voyageur, and studied the physiology of the gastric digestion. There are many pages devoted to the pioneer physicians, their hardships and experiences. One of the most interesting chapters is that by Dr. J. H. Dempster on Medical Education. The first medical college in Michigan was the University of Michigan Medical School, founded in 1850, and now one of the great medical schools in the United States. Dr. Walter H. Sawyer presents a valuable contribution on Medical Journalism. The subject of Prevailing Diseases and Epidemics is well covered by Dr. C. B. Burr and the volume concludes with an excellent summary of Public Health Work in Michigan by Dr. Guy L. Kiefer.

The book is copiously illustrated with well chosen pictures. It is a pity that so large a volume, 829 pages, should not contain an index. We suppose that one will be provided in a future volume, but it would have been more convenient had it been placed in this one.

Francis R. Packard.

Injuries to Joints. By Sir Robert Jones, Bart., K.B.E., C.B., CH.M. (Liverpool), F.R.C.S. (England, Ireland, and Edinburgh), F.A.C.S. (U. S. A.) Ed. 3, 196 pp., 29 illus., Oxford Univ. Press, 1930.

The third edition of Jones' "Injuries to Joints," begun as a war-time manual, fully maintains the standards set by the first two editions. It is to be regretted that the distinguished author does not enlarge the book so as to make it a complete text on the subject.

Physical Diagnosis. By Richard C. Cabot, M.D. Ed. 10, Revised and enlarged. 552 pp., 6 plates, 279 figures. New York, Wm. Wood, 1930.

Any book that runs through ten editions in twenty-five years may be considered as having made its definite place in medical literature, and this statement applies with unusual force to the book before us. In this edition, according to the author, "The more important new matter introduced relates to coronary disease, electrocardiography, cancer of the lung, cardiac asthma, toxic hepatitis, and encephalitis lethargica." Thoroughly up-to-date, Cabot's "Physical Diagnosis" may not only be freely recommended to students as a standard and thoroughly complete textbook on the subject but will be found valuable in the library of every physician and surgeon as a book for continual reference. Well written, well printed, concise and up-to-date, the book has made its place as one of the classics of American medical literature.

Gonococcal Infection in the Male. By A. L. Wolbarst, M.D. 297 pp., 133 illus. 7 col. plates, St. Louis, C. V. Mosby, 1930.

There have been several new books on gonorrhea in the last few years, all of them up-to-date and practical. This is the second edition of Dr. Wolbarst's book the first having been published in 1927. It is unfortunate that, as in the case of so many monographs, this book will be found to be too detailed for the general practitioner and not detailed enough for the specialist.

Manual of Physiology for Students and Practitioners. By H. Willoughby Lyle, M.D., B.S. (Lond.), F.R.C.S., (Eng.) and David De Souza, M.D., D.SC. (Lond.), F.R.C.P. (Lond.) 836 pp., 3 plates, 138 figs., London, Oxford Univ. Press, 1930.

For the third time since 1911 this book has been revised and brought up-to-date. In handy format, tersely written and well printed, this work makes a convenient and practical text and reference work on modern Physiology. By the use of a large type page, small type face and thin paper, the publishers have succeeded in putting a great amount of material into convenient compass and the authors have the happy faculty of eliminating the unessential, thereby making every word and page count.

A CLINICAL STUDY OF THE ABDOMINAL CAVITY AND PERITONEUM

EDWARD M. LIVINGSTON, M.D.

PUBLISHED SERIALLY IN

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CONTENTS

This Number

	Page	A. J. S. Page
SECTION II. THE GASTROINTESTINAL TRACT (CONTINUED)	366	582
B. Anatomical and Physiological Divisions of the Gastrointestinal Tract (Continued)	366	582
Questionnaire	414	630
References	417	633

Previously Issued

		Vol. VIII
INTRODUCTION	1	195
SECTION I. THE CAVITY	6	200, 460 695, 912, 1110, 1325
SECTION II. THE GASTROINTESTINAL TRACT	239	1333
		Vol. IX
	320	366

[In the following pages the Journal page number will be found at the bottom of the page.]

A CLINICAL STUDY OF THE ABDOMINAL CAVITY AND PERITONEUM

Section II. The Gastrointestinal Tract

B. Anatomical and Physiological Divisions of the Gastrointestinal Tract (*Continued*)*

"Nature," it has been asserted, "is interested in function rather than in appearances, in physiology rather than anatomy." And the value and effectiveness of surgical measures are to be judged largely upon the basis of functional end-results. It is insufficient that the surgeon think in terms of anatomy alone. In creating an intestinal anastomosis, for example, the facility and convenience with which two specific bowel segments may be approximated sometimes prove to be a consideration of less importance to the best interests of the patient, than the question as to whether it is this particular segmental union, or some other, which will most completely preserve or restore normal alimentation. In the realm of abdominal diagnosis, also, the interpretation of signs and symptoms rests to a considerable extent upon an adequate understanding of the subject of gastrointestinal physiology. Study of anatomical units of the alimentary canal with their purely arbitrary limits, (stomach, duodenum, ileum, jejunum, colon) reveals little to suggest the identity and location of functional divisions of the tract. And these, therefore, require separate review.

There are three major processes of alimentation, *viz.*, secretion, absorption, and excretion; the great functional divisions of gastrointestinal tract likewise are three in number. These are determined by the manner of distribution of alimentary blood

* Previous installments of this book appeared as follows: Vol. viii: January issue, p. 193; February issue, p. 459; March issue, p. 693; April issue, p. 911; May issue, p. 1109; June issue, p. 1325. Vol. ix: July issue, p. 157; August issue, p. 365.

vessels, lymphatics and nerves. Physiological divisions, for this reason, are fundamental and basic in character. The names of these functional units are based upon the portions of the embryonic alimentary tube from which they arise (foregut, midgut, hindgut). The functional foregut of the adult (secretory division) terminates within the duodenum immediately below the site of entrance of the biliary and pancreatic ducts (ampulla of Vater). The termination of the functional midgut (absorptive division) lies within the left half of the transverse colon near the splenic or left colic flexure. Differences as to the location of anatomical and physiological divisions of the tract are, then, most clearly to be noted in the duodenum and transverse colon. The duodenum proximal to the entrance of the common bile duct is so similar in behavior to the pyloric end of the stomach that the two have been considered, both by surgeons and radiographers, as related.[169] The distal part of the duodenum, on the other hand, falls within a different physiological division. Points which illustrate the practical significance of functional variations within different parts of the duodenum will be dealt with later. Again, the transverse colon, extending between the right and left colic flexures displays, at its extremities, physiological differences which are so profound that it cannot be considered as a single functional unit.[170]

Functional vs. Anatomical Divisions

Visualization of these functional divisions is aided by reference to that stage of development in which the archenteron is still open in its mid-third into the yolk sac (Fig. 141.) The foregut is that portion of the canal situated cephalad to this yolk sac opening (parts cephalad to mid-duodenum); and the hindgut is the portion caudad to the yolk sac opening (all parts distal to and including the left colic flexure).

Embryological Basis for Divisions

The three functional divisions, it may be observed, receive their blood supply from separate sources. The foregut derivatives are nourished through the celiac axis; midgut derivatives are supplied by the superior mesenteric artery, and structures derived from the hindgut are furnished arterial

Vascular and Nervous Supplies

blood by way of the inferior mesenteric artery. The sympathetic nervous system sends fibers to all parts of the gastrointestinal tract, but the supply from the autonomic

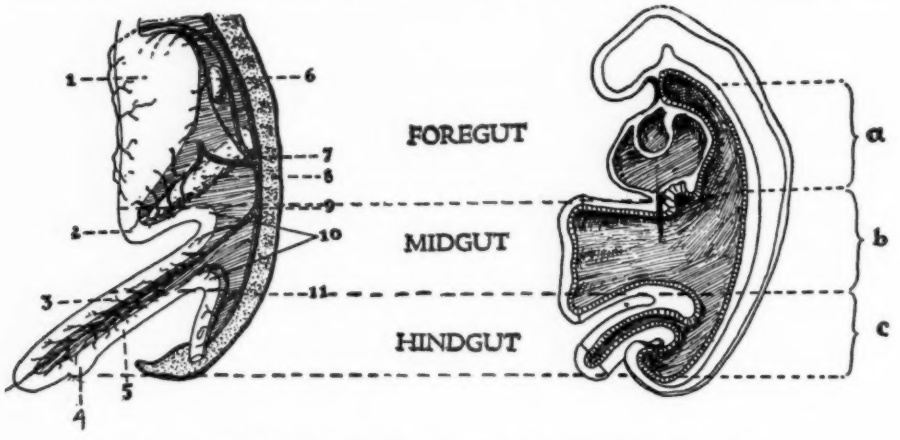


FIG. 141. The functional divisions of the gastrointestinal tract.

a. Foregut. b. Midgut. c. Hindgut.

1. Stomach. 2. Duodenum. 3. Descending limb of intestinal loop. 4. Cecal bud. 5. Ascending limb of loop. 6. Spleen. 8. Pancreas. 9. Superior mesenteric artery. 10. Duodenocolic isthmus or base of loop. 11. Inferior mesenteric artery.

system differs with the different functional units. The foregut and in part the midgut receive fibers from the bulbar autonomics (vagi) while the hindgut receives fibers from the sacral autonomic trunks (nerves erigentes or pelvic vagi).

Surgical Physiology

If the functional divisions are less frequently referred to in surgical literature or seem less perfectly understood, it is probably because as has often been reiterated "operators are on the whole far better anatomists than physiologists." Much of surgical literature falls within the realm of applied anatomy. And it is, perhaps, a fair generalization that anatomy, the older subject, is the more fully presented as to its surgical aspects. Anatomical literature, also, seems to posses a certain precision which appears yet wanting, at times, within the sphere of physiology. Compare for example a description of a vagus nerve with a discussion of vagus functions. But to the truism that the surgeon must "think anatomically" might well be added the admonition that he "act physiologically."

And time spent in study of gastrointestinal physiology is amply repaid both in terms of improved surgical end-results and of increased clinical acumen.

The present review of gastrointestinal physiology will adhere to the previously adopted method of giving an admixture of basic with practical topics. The aim is a correlation of physiological facts with everyday surgical matters. The abrupt transition from one practical topic to another, an inherent shortcoming of this approach to the subject, is compensated for, to a degree, by the headings given individual paragraphs.

Gastric Absorption

As might be anticipated, because of the overlapping of nerve fibers and the anastomosis of blood vessels at divisional boundaries, functional activities extend somewhat beyond general territorial limits. Thus within the stomach (secretory division) there is chiefly an outpouring from the mucous membrane of gastric juice with its hydrochloric acid, pepsin, rennin, lipase and mucin; but to a limited degree absorption also takes place from this viscus. Gastric absorption, however, is so slight that if foods reach the stomach alone, starvation soon results. And the surgeon knows that when he deals with an obstruction to the pylorus which is becoming complete, it is essential that some method such as a gastric resection, gastroenterostomy or jejunostomy, be employed to allow nourishment to reach the functional midgut. The mucosa of the stomach absorbs practically nothing but limited amounts of alcohol, simple sugars and certain of the mineral salts.[171] Even water, which normally passes at once to the duodenum along the lesser curvature (magenstrasse or canalis gastricus) is not absorbed by the gastric mucosa.[172]

Absorption in the Distal Colon

The distal colon or expulsive division of the bowel (descending, iliac, pelvic colon) similarly, has little absorptive power. This terminal third of the gastrointestinal tract is to be considered as chiefly a storehouse to accommodate feces until a convenient time for their evacuation.[173] Unaltered proteins are not taken up by the colic mucosa and fats remain unabsorbed.

The administration of so-called "nutrient enemata" of eggs, beef juice, cream, etc., seem, accordingly, irrational. Of nutritive substances monosaccharides alone are absorbed. And it is even stated that about 80 per cent glucose remains within the colic lumen.[174] Water and sodium chloride may be introduced by rectum (Murphy drip; Harris drip; rectal infusion; proctoclysis); but in calculating the patient's nutritive and fluid balance the surgeon must take into account the limitations and uncertainties of colic absorption. Sugar solutions administered in high concentrations (i.e. above from 3 to 5 per cent) may so irritate the rectum that the patient cannot retain the substance. Thus the possibilities of rectal feeding are slight; obviously the function of the terminal colon is almost exclusively excretory or expulsive. And secretory activity within this portion of the bowel is limited to the discharge of mucus, that natural lubricant essential to normal defecation.[173]

Introduction of Fluids with Bowel Stasis

With adynamic ileus (bowel paresis or paralysis) the surgeon finds himself unable to administer desired amounts of fluids or nourishment since the stomach and colon (secretory and expulsive divisions) are unable to provide the requisite absorption. Hypodermoclysis or direct infusion of saline or glucose must then be resorted to. Postoperative intestinal stasis may be anticipated by having the patient swallow a duodenal tube before operation. Then fluids may be placed at will directly into the absorptive part of the alimentary canal. When bowel stasis is marked, however, and the absorptive powers of the mucosa are severely impaired (edema, vascular occlusions, lowered portal circulatory pressure) water and nourishment must be administered by other routes. It is of historical interest to note that prior to the period of asepsis the surgeon was incapable of safely administering liquids by any avenue other than through the alimentary canal (mouth or rectum); and not the least benefit or boon of asepsis is the ability to secure absolute sterility of fluids to be placed beneath the skin, into the peritoneal cavity, or into the circulation for the combat of shock and dehydration.

Physiological Compensatory Mechanisms

There has been provided to a remarkable degree a compensatory mechanism whereby in certain abnormal states one functional bowel division may assume to a far greater extent

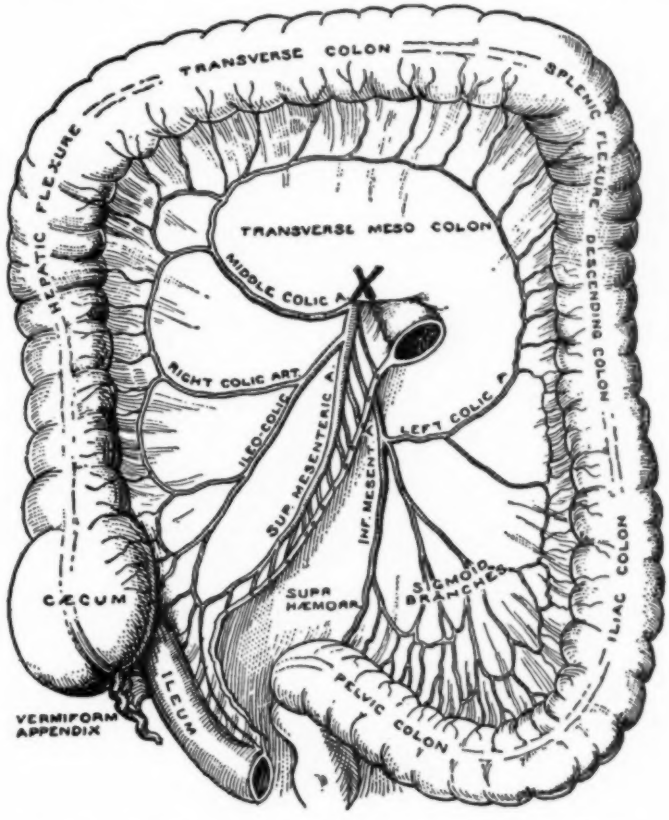


FIG. 142. Embolic plugging of the superior mesenteric artery.

A. Demonstrates how complete stoppage of the superior mesenteric artery (point x) causes gangrene of intestine from the proximal jejunum to the transverse colon. Loops of small intestine have been removed to show the colic circulation but their arterial supply may be seen within the root of the mesentery. (From Taylor's Operative Surgery.)

than usual the functions of another. Cases are reported in which almost all of small bowel (absorptive area) has been resected yet the patients survive over periods of months or years.[175] Such extensive resections have been made as a desperate necessity after embolic plugging of the superior mesenteric artery with resulting jejunoileocolic gangrene (Fig. 142). Beginning within healthy bowel distal to the area of gangrene

the surgeon has incised the mesentery along most of its 6 in. base, thereby freeing loop after loop of small intestine until healthy gut proximal to that found black was encountered.

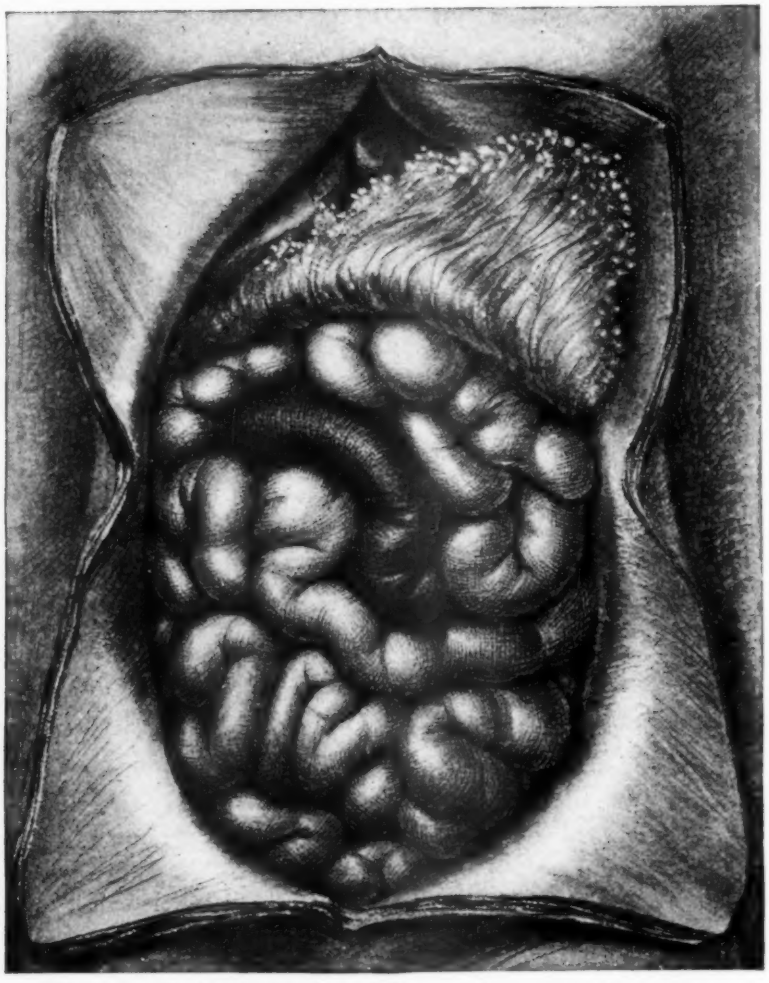


FIG. 142. Embolic plugging of the superior mesenteric artery.

B. Shows gangrene of a single loop of intestine from embolic plugging of a small branch of the superior mesenteric artery. (From Eisendrath's Abdominal Diagnosis. W. B. Saunders & Co.)

The subsequent anastomosis of the jejunal stump to the ascending or transverse colon has left the patient with only those portions of the gastrointestinal canal supplied by the celiac axis and the inferior mesenteric artery (i.e. the func-

tional secretory and expulsive divisions). The remarkable recoveries which have followed such removal of over 15 ft. of intestine well illustrate a great compensatory or adaptive mechanism. Notwithstanding, however, both slight natural overlapping of functions and such compensatory activities as observed in abnormal states the three main functional processes of alimentation are carried on to a major degree within the three great functional divisions of the gastro-intestinal canal.

The practical significance of physiological divisions will be further illustrated by certain points relative to intestinal fistulae and stomata.

High vs. Low Jejunal Fistulae

From a high jejunostomy there is a copious and almost continuous outpouring of liquid which is highly irritating to tissues of the body wall. This substance (chyme) reaches the surface before traversing the absorptive area of the bowel and a marked loss from the system of nutriment consequently results. Clinically the distressing signs and symptoms of dehydration and starvation follow. A high fistula shows little tendency to spontaneous closure and unless it be dealt with by secondary operation will continue to discharge, if the patient survives, for from six to nine months, or perhaps permanently.[176] Thus although a high jejunostomy for intestinal obstruction may relieve bowel distention and rid the patient of material of marked toxicity, proving at times a life-saving measure, the advantages may be largely abnegated by disagreeable and dangerous late effects unless the jejunostomy is promptly closed by the surgeon after its purpose has been fulfilled. Low bowel fistulae produce less dehydration and starvation, their discharges are intermittent and prove less irritating, and they exhibit a definite tendency to close spontaneously.

Feeding Intestinal Enzymes

Surface excoriations and digestive changes in the belly walls about jejunal stomata have been dealt with satisfactorily through furnishing the pancreatic and intestinal enzymes, (trypsin, erepsin, amylopsin, maltose, lactose, lipose) suf-

ficient food upon which to act. This "feeding of the ferments" in order to spare the patient's own tissues has been accomplished both from within and from without. A diet rich in eggs, for example, has effectively stopped the proteolytic changes in living structures.[177] Again, the desired effect has been produced by saturating the surgical dressings with kaolin,[178,179] or by beef juice; also by applying steak to the surface. Weak hydrochloric acid added to the protein used in the dressings serves to neutralize excessive alkalinity of the succus entericus. So characteristic of high intestinal fistulae is the presence of deep-seated adjacent excoriation and tissue digestion that the surgeon can usually tell by inspecting the wound, when a fistula which he is examining for the first time is of jejunum and when of colon or some low segment of bowel. Colic fistulae are as a rule free from severe surrounding irritation.

Diet with Artificial Ani and Fistulae

For a patient having an artificial anus or enteral fistula diet plays a rôle of considerable importance in determining postoperative comfort.[174] A patient who is observant, after study of his stoma, can soon know with fair accuracy what foods he may safely ingest. In general the diet should contain little that is bulky or leaves a quantity of residue. Raw foods tend to be acted upon only low in the gastrointestinal tract for cellulose coverings are not dissolved by the enzymes but rather are broken up by the action of bacteria and by putrefactive changes in the colon. Naturally such foods are disadvantageous with fistulae. Cathartics and cathartic foods (figs, prunes, baked apples, bran, cereals) tend to cause discomfort with high fistulae. Simple sugars, eggs, meat juices, milk and milk products are among the most acceptable foods in most cases. With high jejunal stomata, as stated, a diet rich in proteins diminishes the irritating qualities of the fistulous discharges.

Mucous Fistulae

Feces is not as the layman usually believes chiefly composed of undigested residue from ingested foods. The greatest bulk is formed of waste products of the intestinal canal and its deriv-

atives (secretions and excretions) plus bacteria; food remains constitute but a small fraction of this alvine discharge.[180] The postoperative patient, though receiving no food or having an exceedingly limited intake still forms feces which needs elimination and this substance does not vary greatly from that produced during health. Any moderate increase during health, as is shown by stool examinations, is due less to undigested food materials than to the degree of activity of liver, pancreas and glands along the intestinal canal which activity tends to be greater in the normal healthy individual than in one confined to bed. These facts relative to feces and the output of the alimentary mucosa make it clear that a loop of bowel which is closed at both ends, hence isolated from the alimentary current, even though rendered functionally inactive, cannot safely be returned to the abdomen. If for any reason such isolated intestine cannot be resected it must be opened and a permanent communication made with the surface.[181] Such a stoma is referred to as a mucous fistula. As functional demands are withdrawn from this isolated bowel its mucosa gradually degenerates and atrophies and the discharge from the mucous surface necessarily diminishes; yet an occlusion of the external opening ensures an ever increasing distention of the loop which invites disaster. A completely isolated loop, open at each extremity with a mucous stoma, is referred to as a "Thiry-Vella fistula."[182] (Fig. 143.)

It is the custom of some operators when attempting an appendectomy in a difficult case to work from the base of the appendix toward its tip, rather than the reverse. In the most exceptional of instances after the appendix has been severed and the cecal stump inverted, it is found almost impossible or quite unsafe to remove the deeply situated and adherent tip of the appendix. Here the possible expedient might well be employed of attaching the base of the severed appendix to the skin wound as a short mucous fistula, rather than to leave the diseased organ entirely within the peritoneal cavity.

A. J. S., n. s. Vol. IX, No. 3, p. 591

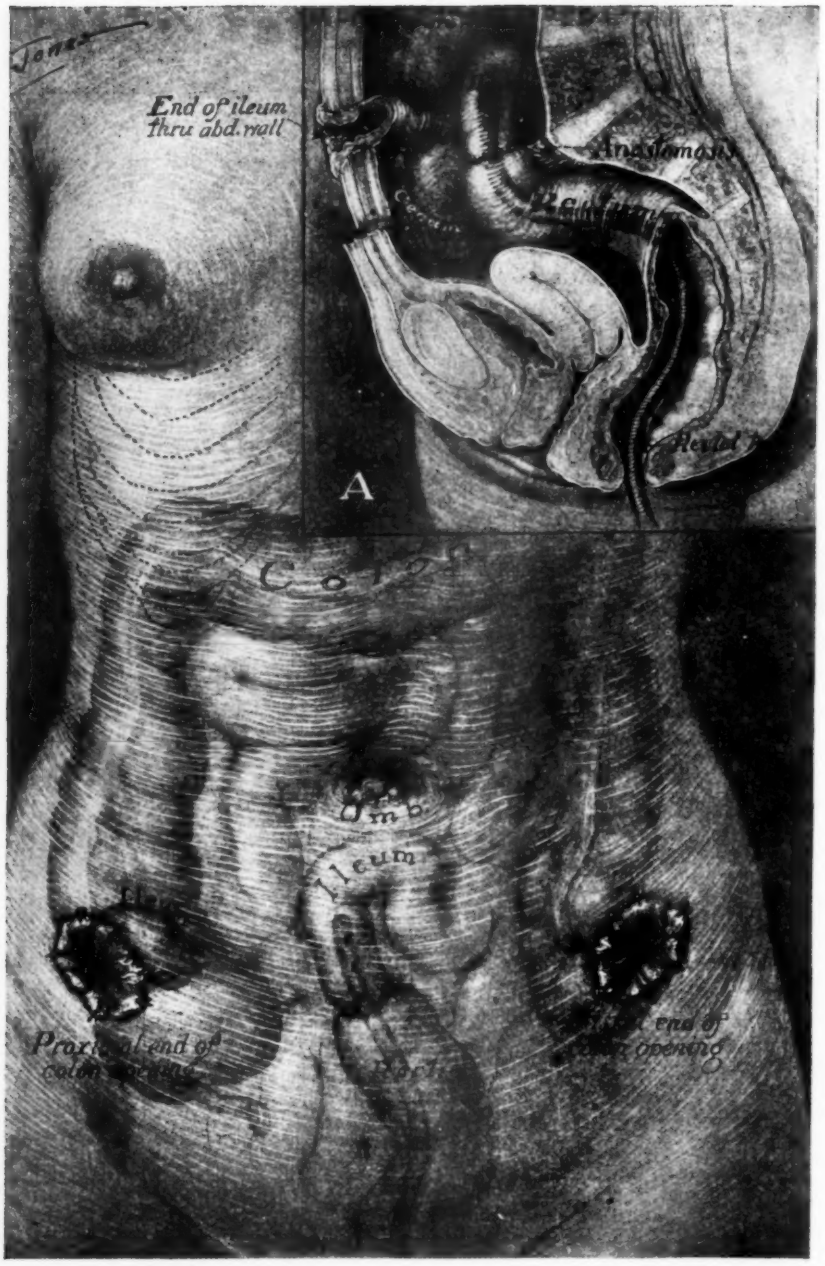


FIG. 143. Operation for the elimination of the ascending, transverse and descending colon; intestinal exclusion. The ileum has been severed 10 cm. from the cecum and the cecal end passed through a McBurney incision, making a permanent colostomy at this point. The sigmoid has also been severed and the end of the descending colon passed through a similar McBurney incision on the left side. An end-to-end anastomosis was then made between the proximal end of the ileum and distal end of the sigmoid, and a rectal drainage tube passed up into the ileum. The colon can now be flushed out through its two colostomy openings. (From Ochsner and Percy's A Manual of Surgery.)

A Temporary vs. a Permanent Artificial Anus

In creating a temporary stoma in the treatment of paralytic ileus caused by acute peritonitis one does not hesitate, with the patient in a critical condition, to utilize any presenting loop of bowel in preference to making an extensive search for a more desirable segment. This permission to "use any loop" should not, however, be extended to situations where permanent artificial ani are being created. Here, other things being equal, that portion of gut should be utilized which will leave the patient with as much normally functioning bowel as is feasible. To employ small bowel in making a permanent anus throws the entire colon permanently out of function and results in the constant drizzling of nutritive chyme as described under the heading of jejunal fistulae. When the descending or sigmoid colon is selected for a permanent anus the outflow of feces is intermittent and from the excreta all food value has been removed; hence no marked starvation or dehydration ensues. For a permanent artificial anus the terminal functional third of bowel represents the site of election.[183] To use a midgut derivative places a distinct drain upon the patient's nutritional balance and to use a foregut derivative for this purpose makes quite certain a relatively short life expectancy. It is true that the variety of conditions which call for artificial ani make it necessary to individualize and to arrive at the decision as to the exact site for the stoma only after taking into consideration all of the factors involved; the pathological necessities of the case, matters of anatomical convenience, of the appearance and comfort of the patient, but above all of the functional end-result.

Intestinal Exclusion or Segregation

Generalizations similar to the foregoing apply with equal aptitude to intestinal short circuits or exclusions. By an "exclusion" of the bowel is meant the surgical procedure for rendering any portion of the intestinal canal functionally inactive; the segment thus eliminated by the short circuit is said to be "excluded." Such an operation may be necessitated by the presence of a growth which cannot be removed, in which case the portion associated with the tumor is excluded and the

intestinal current thus diverted from the diseased area. A portion of bowel containing a fistula may be excluded (for example a tuberculous loop containing a spontaneously created

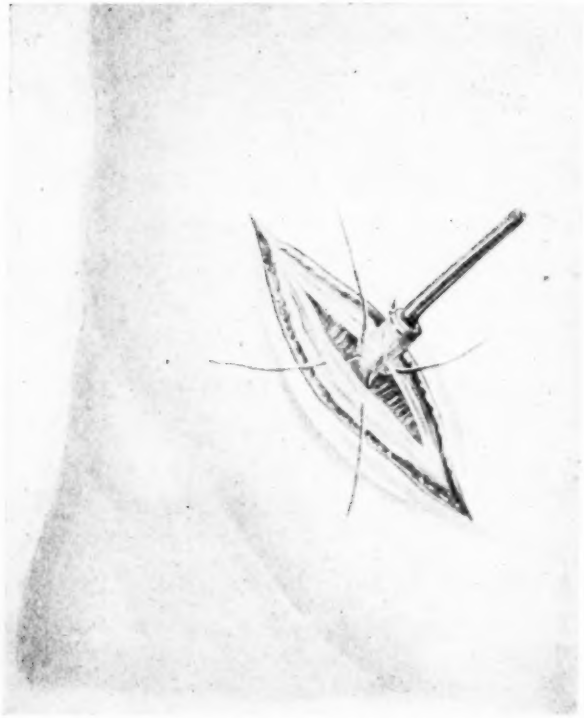


FIG. 144. Appendicostomy. The incision in the skin is made long to show the details of the operation. It need not be longer than 1 to 1½ inches. (From Moynihan's Abdominal Operations. W. B. Saunders & Co.)

sinus) in order that the stoma may close and the diseased bowel heal. The basic principle is to exclude as little of the alimentary canal as is consistent with the pathological condition being dealt with, and to preserve as much as possible of each of the functional thirds of the tract. Thus to short circuit about an irremovable neoplasm at the splenic flexure (left colic flexure) a colo-colic anastomosis (joining transverse colon to sigmoid or pelvic colon) is preferable from the standpoint of functional considerations to an ileosigmoidostomy.[184]

Preliminary Stomata

A preliminary stoma or fistula is sometimes required as part of a two-stage or multiple-stage operation. In the presence

of an obstruction low in the colon a preliminary colostomy, cecostomy or ileostomy allows the patient to be treated until the systemic effects of the bowel stasis present have been relieved. Colic resections performed in the face of acute obstruction are almost always fatal, hence "with acute colic obstruction temporary stoma production becomes an imperative necessity." To create a cecostomy or appendicostomy is a simple procedure which can be performed under local anesthesia (Fig. 144). At the time of colic resection (second stage) such a fistula or enterostomy above (cephalad to the site of anastomosis) relieves postoperative tension at the anastomotic suture line. Of all the precautions necessary to success with resections of the colon (prevention of contamination at time of operation; provision by suitable drains for possible leakage; peritonealization at suture line, etc.) none is of greater importance than the prevention of tension after the anastomosis by means of such an associated fistula higher in the gastrointestinal tract.

Early Closure of Temporary Fistulae

Spontaneous closure of temporary fistulae is prevented by bowel obstructions distal to the stoma and by eversion of the mucous membrane. (Fig. 145 C and F.) Thus to ensure prompt closure of a fistula designed for but temporary aid in the functioning of the bowel it is necessary to see that stenosis beyond the stoma has been relieved. But in addition early closure may be aided through attaching only a small part of the anti-mesenteric border of bowel to the body surface, or through using a small caliber tube buried within the bowel wall after the Witzel method and passing this tube on its way to the surface through a hole created for the purpose within the great omentum (Fig. 146). The latter method prevents pouting of mucous membrane and it is quite certain that if there is no bowel obstruction remaining beyond the fistula it will close promptly upon the withdrawal of the tube, the omentum acting as a plug.

Additional illustrations of the practical significance of bowel divisions, both anatomical and physiological, will be

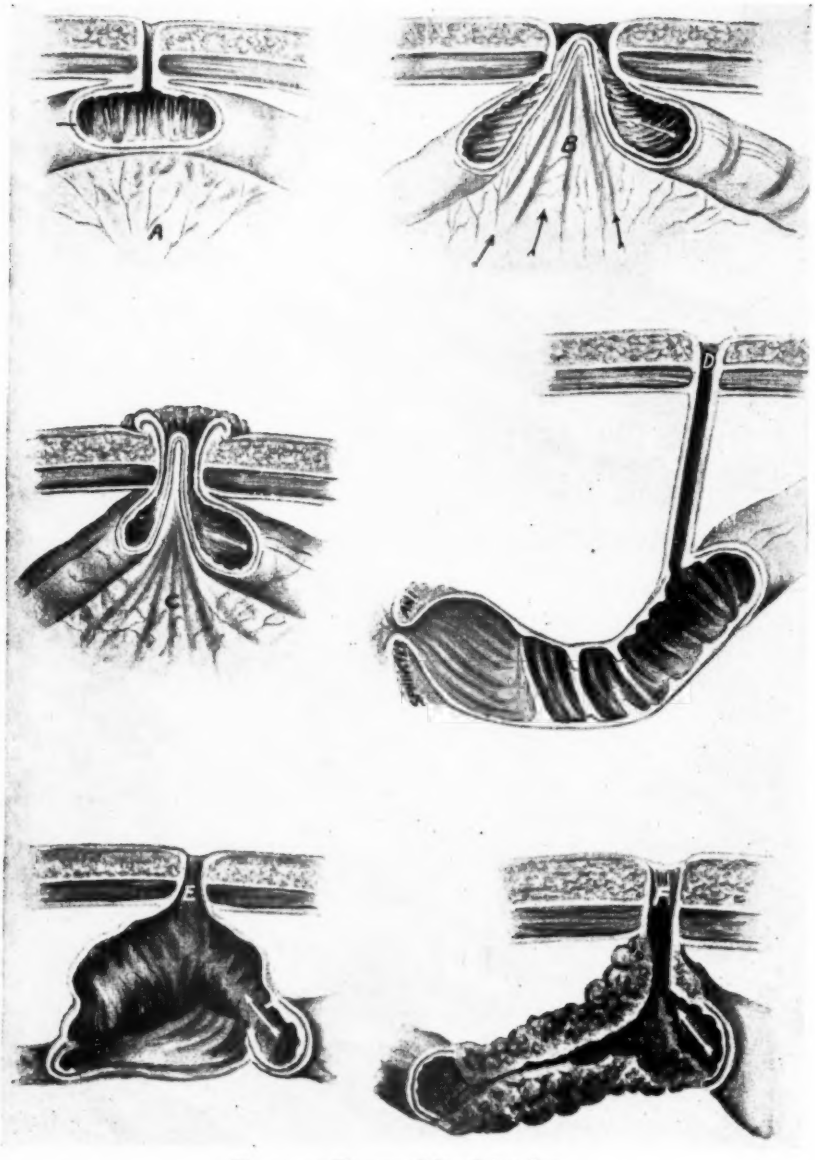


FIG. 145. Types of fecal fistulae.

A. Simplest form. B. Spur formation or reducible hernia type. C. Artificial anus or irreducible hernia. D. Fistula leading to intestine remote from abdominal wall. E. Fistula leading to large cavity resulting from gangrene due to mesenteric embolism. F. Fistula leading to intestine above cancer. (Coffey.)

furnished by a brief review, from a clinical standpoint of certain important intestinal diseases.

There are but three common surgical diseases of bowel

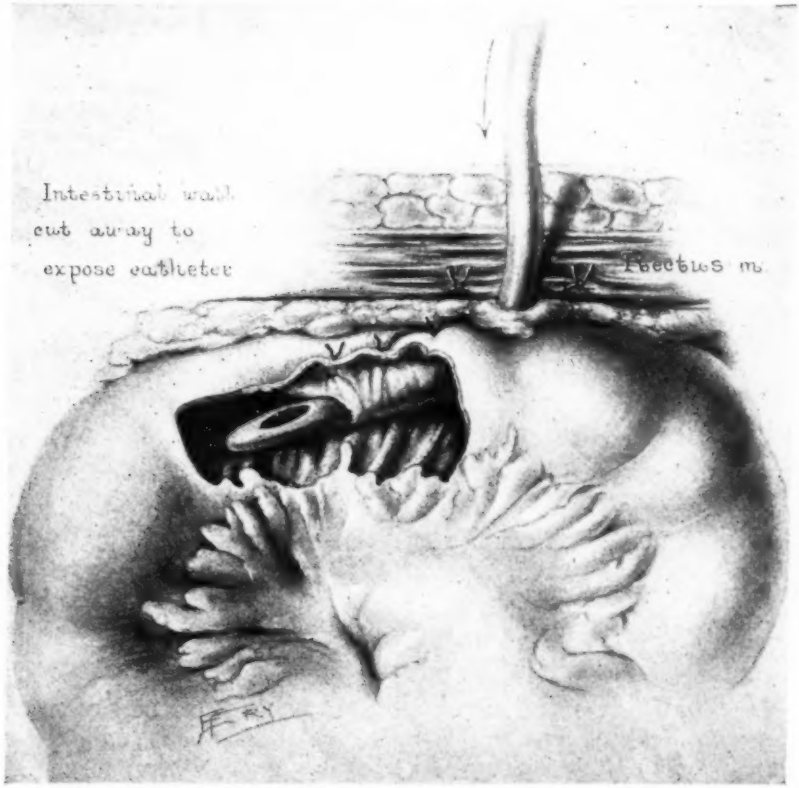


FIG. 146. Tube enfolded in the wall of the bowel, passing through the omentum and the abdominal wall. (After W. J. Mayo.)

between duodenum and rectum. These are (1) appendicitis (2) acute intestinal obstruction and (3) neoplasms. Hospital records show that over 90 per cent of signs and symptoms with proved surgical disorders of this portion of bowel (from duodenum to rectum) arise from one of these three conditions.[185] Acute intestinal obstruction, affecting as it does all parts of the bowel, serves quite ideally to illustrate differences in function at various levels within the alimentary canal.

Common Surgical Diseases of Intestine

Intestinal obstruction is classified in many ways; but to the clinician the most important is according to the site at

Clinical Varieties of Intestinal Blockage

which the intestine is blocked. Complete bowel stoppage, if unrelieved, terminates fatally but the fatal outcome is much more prompt when the point of obstruction is high in the canal. Not only does the site determine in large measure the potentialities of the condition but also the character of the picture observed at bedside examination and the rate of progress of the disease (fulminating course; delayed course). Accordingly intestinal obstruction of the mechanical type is classified clinically as (a) high obstruction and (b) low obstruction. The site for incision, the prognosis to be given, and the type of after-treatment to be employed depend upon ascertaining, if possible, the site at which obstruction has occurred.

Causes of Death with Intestinal Obstruction

A fatal outcome with intestinal obstruction is often due to a terminal and associated peritonitis; but the more immediate causes of death are (1) loss of fluid from the body (2) disturbance of the acid-base equilibrium, due to rapid elimination of substances contained within the lost fluid, and (3) a toxemia, apparently caused by the absorption of highly toxic material known to exist within the lumen of the obstructed bowel.[186] The higher the site of obstruction, the more prominent are the latter factors and the more rapid and certain the fatal issue. The reasons for this fact involve basic considerations regarding physiological chemistry and functional variations throughout the alimentary tract. These will be taken up for review after other clinical and diagnostic points have been dealt with. And statements as to specific mortality rates, both with high and low types of obstructions, with and without operations, will be reserved for subsequent paragraphs which have to do with treatment and aftercare of this important surgical disorder.

Types of Vomiting

It is by persistent vomiting, a cardinal sign of intestinal obstruction, that such large quantities of water and essential substances leave the body, causing the dehydration and in part the disturbance of the acid-base equilibrium. With duodenal and upper jejunal obstructions vomiting tends to begin early, to be almost continuous, and to be copious in amount; the patient may die within a relatively few hours, the con-

dition running a fulminating course with a rapid loss first of gastric contents, then of mucus and bile, and finally of a brownish fluid having a fecal odor. The vomitus may seem

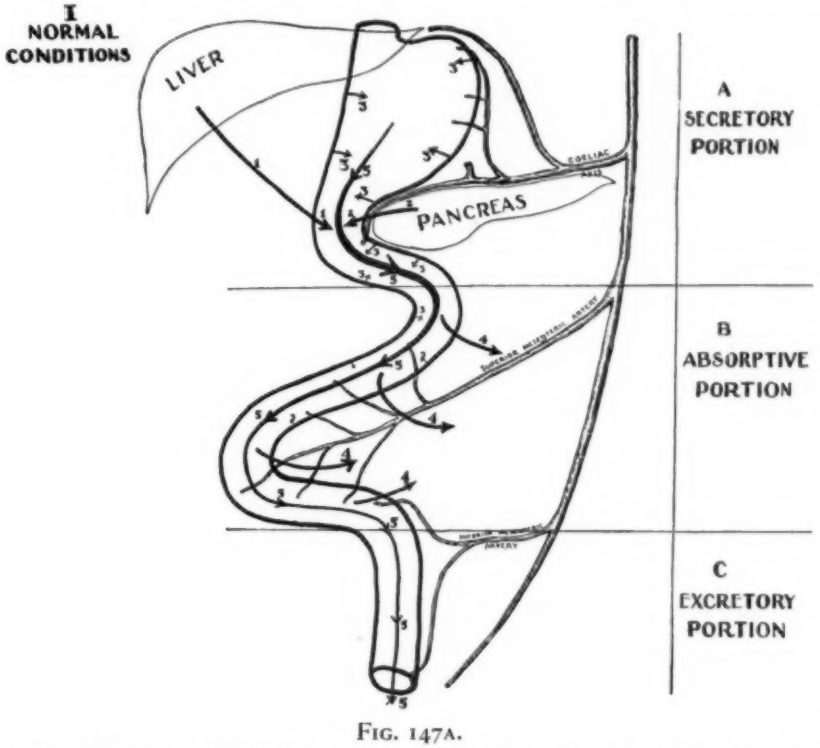


FIG. 147A.

1. The inflow (1, 2, 3) is balanced by intestinal absorption (4) and the small outflow (5). 1. Biliary inflow. 2. Pancreatic inflow. 3. Gastrointestinal outflow. 4. Intestinal outflow. 5. Anal outflow.

to well-up from below repeatedly and continuously in mouthful amounts as though simply representing an overflow, unassociated with nausea or effort on the part of the patient (regurgitant vomiting). This may occur when no food is being ingested or water taken by mouth. An explanation of this clinical observation is the following: with high obstructions the bowel above the blockage is being rapidly filled with fluids which enter the lumen by way of the mucosa, since secretion is the function of foregut derivatives; (it is stated that the amount of liquid secreted into the intestinal lumen within a period of twenty-four hours more than equals the total bulk of

the blood and lymph of the body);[187] the fluid cannot pass downward to be absorbed as it would under normal conditions and the only outlet for the overloaded bowel is into the stomach

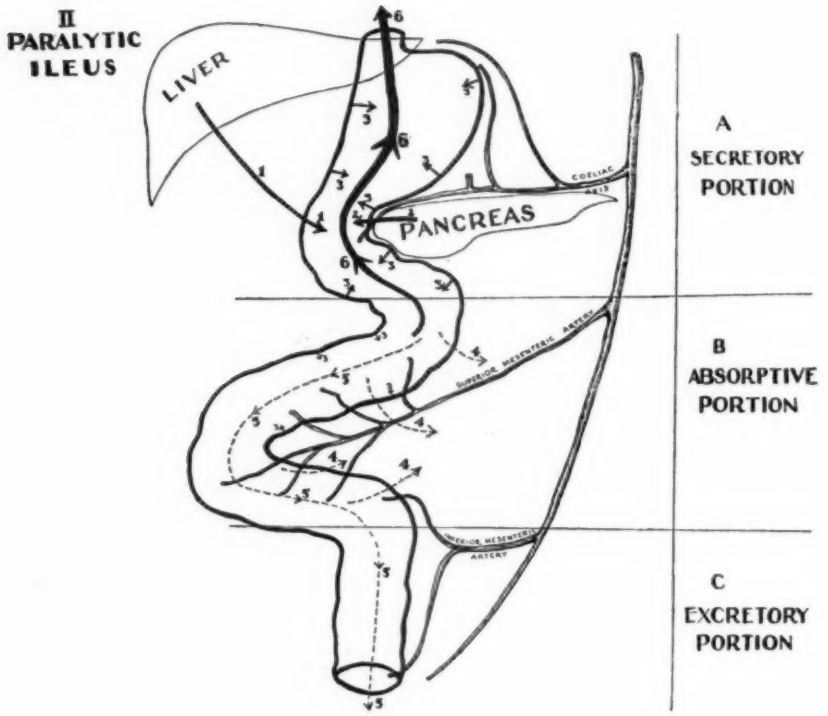


FIG. 147B.

II. Paralysis of the intestinal musculature is followed by marked abdominal distention followed by overflow or regurgitant vomiting (6). Intestinal absorption (4) is greatly diminished and nothing is passed by anus (5). Compare type of distention with that of high mechanical obstruction. 1. Biliary inflow. 2. Pancreatic inflow. 3. Gastrointestinal inflow. 4. Intestinal absorption. 5. Anal discharge. 6. Buccal outflow.

and out through the mouth (Fig. 147, II). Upon the other hand, with acute obstructions low in the canal (colon) vomiting tends to be delayed, scanty or absent. There may be a single siezure of vomiting such as occurs with any severe intro-abdominal insult (initial vomiting), but the obstruction may be complete over a period of many hours or even days without a repetition of the occurrence or the onset of repeated vomiting. Even the preliminary or initial seizure may be absent.

Accumulated material passing toward the stomach and mouth, with low obstructions, must pass through the absorptive area of the bowel, where the amount becomes so greatly reduced

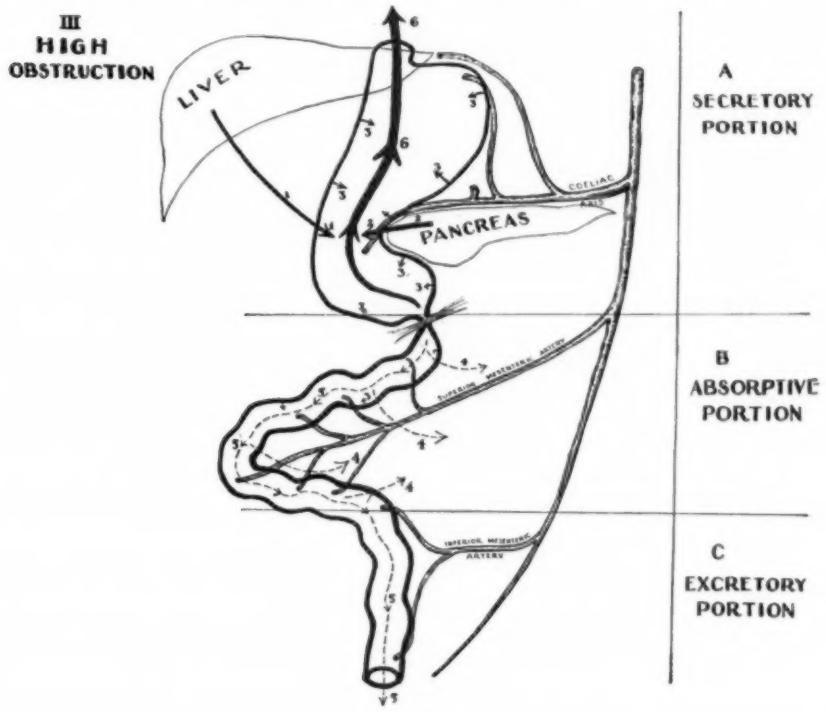


FIG. 147C.

III. Due to blockage proximal to the absorptive area (4) the only outlet for the overloaded stomach and duodenum is by way of the mouth (6).

that the tendency to vomiting is diminished or eliminated (Fig. 147, III).

Fecal Vomiting

Fecal vomiting, as the term implies, means the expulsion of vomits which contains particles of feculent material. Obviously this could be present only with obstruction very low in the intestinal canal, for it is only here that feces is found. Yet clinically vomitus containing true feces is seldom, if ever, observed. And so-called "fecal vomiting" often is merely the condition in which the expelled material is dark in color and has the strong odor typical of colon bacillus infection. Vomiting of the latter type need not imply that the point of obstruction

is low in the canal, for with stasis colon bacilli will invade portions of the bowel normally free from gross contamination with putrefactive organisms, and in obstructed segments

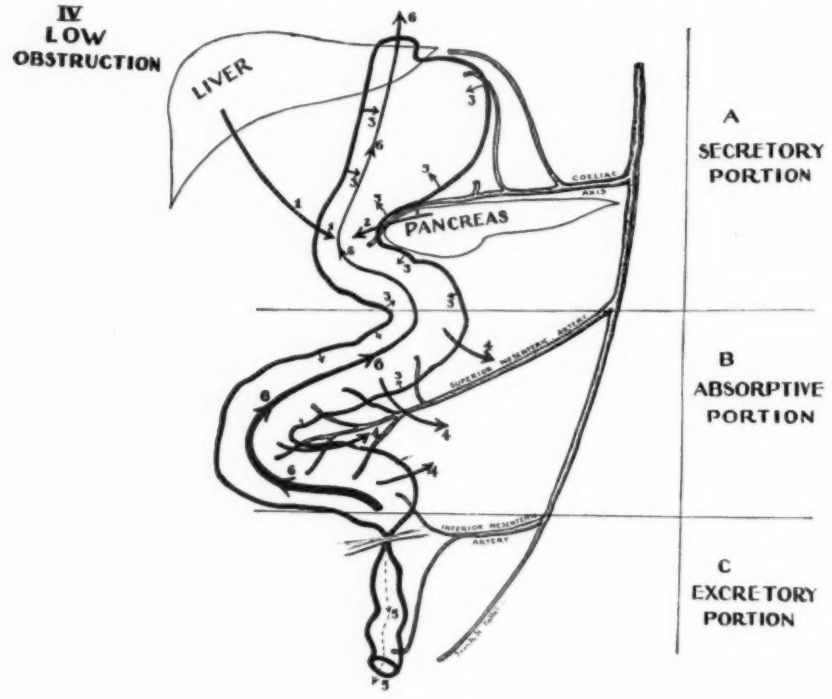


FIG. 147D.

IV. Outlet by way of the anus (5) is blocked but as the intestinal content is forced upward toward the mouth (6) much is absorbed (4), delaying and diminishing the act of vomiting. 1. Biliary inflow. 2. Pancreatic inflow. 3. Gastrointestinal inflow. 4. Absorptive outflow. 5. Anal outflow. 6. Buccal outflow.

bacteria multiply with unusual rapidity causing the changes in the color and odor of the intestinal contents[188] (see work of Cushing on intestinal flora).[189] After an abdominal operation, similarly, where the bowel wall has been found severely damaged, it is not uncommon to observe within from one to three days a wound discharge which is dark and odorous. The tendency is to judge that a fecal fistula has formed, yet the definite opinion that this complication has arisen may well be withheld until true fecal discharges or copious amounts of gas have been noted, for the condition may represent a colon

A. J. S., n. s. Vol. IX, No. 3, p. 602

bacillus peritonitis with resultant discoloration and odor to the discharge, and the leakage may so abruptly terminate as to make it quite certain that no fistula was present.

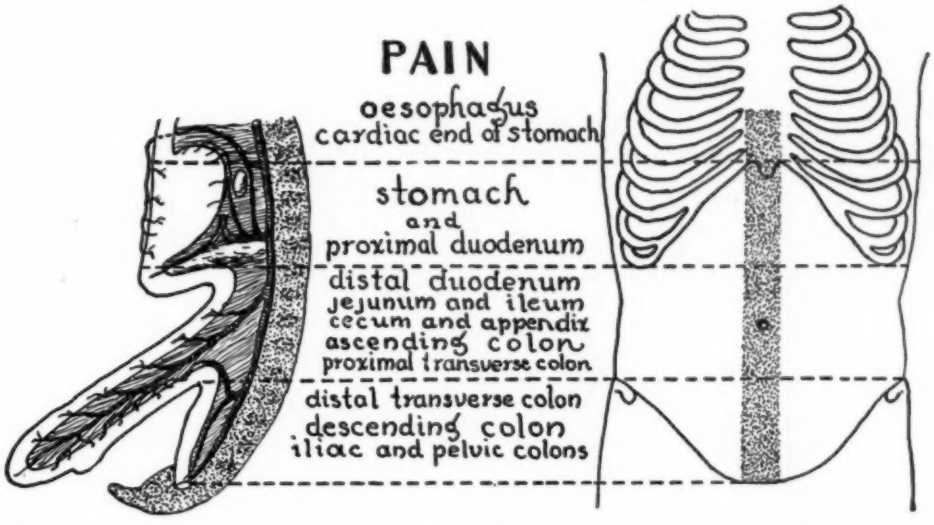


FIG. 148. Study of reference of gastrointestinal tract pains to three equidistant zones in midline of anterior abdominal wall.

Midline Pains with Obstructive Lesions

The site of pain experienced with acute intestinal obstructions serves still further to emphasize the importance of a division of the gastrointestinal tract into thirds. It has been noted in foregoing pages that there are three equidistant zones along the midline of the anterior abdominal wall to which pains arising from affections of the stomach and intestines may be referred (Fig. 148). These surface areas, it was observed, represent the sites for pain reference for the functional thirds of the tract. In the presence of acute obstructions, accordingly, the clinician often is furnished with a physiological guide (pain-reference) as to the location within the canal of the obstructive process. To illustrate: Given a patient complaining of colicky pain localized a short distance above the umbilicus (and associated with other signs and symptoms typical of obstruction) the suggestion is that the jejunum is the bowel segment involved (physiological deduction); and if this patient has also a visible and tympanitic mass within the left hypochondrium, the

evidence supports the supposition that there may be present a herniation into the retroperitoneal tissues through the fossa of Treitz (anatomical deduction).[190] Again, given a patient who for years has been a sufferer from chronic duodenal ulcer but now complains of pain within the hypogastrium, even the past history of a proved ulcer or the persistence of a duodenal cap deformity as demonstrated by roentgenograms does not remove the suspicion that the present complaint arises from some lesion within the colon rather than one within the duodenum,[190] and digital, or instrumental examinations of the rectum and radiographic examinations of the colon are definitely indicated. In ways suggested by such cases anatomical and physiological leads are to be followed and correlated and weighed with other types of diagnostic evidence in attempting to ascertain the presence and nature of gastrointestinal tract affections.

Two Distinct Types of Abdominal Pain

There are two distinct types of severe pain which occur with surgical disorders within the abdomen. With acute intestinal obstructions either or both may be observed. The first type of pain is the typical colic. This comes and goes characteristically and is due, in all probability, to intermittent peristaltic efforts upon the part of the intestine to overcome the resistance at the point of blockage of the bowel (smooth muscle spasm; see biliary colic, renal colic). This pain is usually referred to the midline. By derivation the term colic implies that the seat of the pain is in the colon but this original meaning has been superseded by a broader application of the word. Colicky pains (visceral disturbances) are often associated with sensory, motor and trophic changes, segmental in their distribution, within tissues of the body walls (see visceral neurology). The second type of pain seems deep seated and is more constant or steady in character. It may be referred to any part of the abdomen. This pain is often associated with rebound tenderness and supposedly is due to inflammatory changes in the peritoneum. By rebound tenderness is signified the sudden discomfort experienced by the patient as the examiner's fingers, which

have been pressed deeply and steadily inward toward the peritoneal cavity, are quickly withdrawn. Motor and sensory changes in the body walls, when these are present with the second type of pain, are not segmental or metameric as to distribution, a fact of obvious importance in abdominal differential diagnosis. With acute intestinal obstruction the pain picture varies with many factors, among them, the underlying or primary causative lesion, the type of patient, the condition of the patient at the time of examination (toxemia exhaustion, the recent use of opiates, etc.); this pain picture may rapidly change or shift with progressions or regressions of the pathological process within the abdomen (gangrene, perforation, spreading peritonitis). Of the two types of pain either may be present alone or both may be present simultaneously, sometimes one and sometimes the other predominating. But in general, the typical pain of acute mechanical intestinal obstruction is colicky in character; when a secondary steady pain becomes superimposed it usually implies the onset of a complicating peritonitis due to bowel leakage consequent upon gangreous or other vascular changes in the gut wall at the site of obstruction or within the obstructed loop.

Moving Clinical Pictures

Ingested foods are carried along the alimentary canal by the action of smooth muscles within the walls of the esophagus, stomach, and intestines; digestive juices (ferments, acids, alkalies) are added by the alimentary glands; after selective absorption of nutritive elements into vascular and lymphatic channels has occurred, needless food residues and end-products of the digestive process are, at a convenient time, expelled from the body. This threefold function of the gastrointestinal tract (secretion, absorption, expulsion) abruptly terminates when for any reason (see classification) the bowel becomes acutely obstructed. With complete stoppage neither flatus nor motion is passed spontaneously through the rectum (absolute constipation). Even enemata and irrigations can bring down nothing from above the point of obstruction and after the distal

Four Chief Symptoms of Acute Obstruction

bowel has been washed out, fluids will return clear. The one exception to the rule of absolute constipation is that of intussusception in which bloody and mucous diarrheal discharges arising from invaginated bowel may be passed. As the bowel lumen fills with fluids and gases above the point of obstruction (recall the amount of secretion entering the bowel lumen within twenty-four hours) this upper portion of tract (cephalad) undergoes rapid and steadily increasing distention. With high obstructions, since there is little or no absorptive activity within the cephalad loops, the only outlet is by way of the mouth in the form of repeated emesis and regurgitant vomiting (see Fig. 147). With obstructions well along within the gastrointestinal tract, as the voluminous coils of bowel above the blockage undergo distention, the abdomen as a whole also becomes progressively distended. Reference has already been made to the occasional occurrence of visible ladder patterns from distended small intestinal loops, to the peripheral or horseshoe shaped distention of the abdomen from obstructions in the sigmoid colon or rectum, and to the enormous, generalized, and ever increasing enlargement of the abdomen consequent upon bowel stasis due to paralytic ileus (adynamic intestinal obstruction). The colicky pain, as noted, represents the effort of the intestinal musculature to drive the contents of the bowel lumen beyond the constriction. With abrupt cessation, then, of the processes of alimentation the four most important symptoms are:

(1) colicky pain
(2) regurgitant vomiting
(3) absolute constipation and
(4) steadily increasing distention.

While as will be observed in a consideration of individual types of intestinal obstruction there are instances in which one or more of these cardinal symptoms may be slight in severity or even absent (little or no pain with carcinomatous obstruction; lesser degree of emesis with low obstructions; diarrhea rather than constipation with intussusception; little

generalized distention with very high obstructions) these are nevertheless the outstanding guides in the immediate history which point to the diagnosis of obstruction. And it has been stated as a clinical rule: "If a patient suffering from a sudden attack of abdominal pain has constantly recurring vomiting and every effort to secure the passage of feces or flatus results negatively, a diagnosis of intestinal obstruction may be made."[191]

Other symptoms and systemic signs observed with obstructions have to due largely with the underlying causative lesions such as cancer (cachexia); with the dehydration (thirst, dyspnea); with the excessive stimulation of afferent nerves (shock, rapid pulse); with the toxemia (urinary changes, leucopenia, coated tongue); and with complications such as peritonitis (pulse, temperature, leucocytosis). The late picture with obstruction is as hopeless as it is typical and an early diagnosis, the *sine qua non* of success in treatment, must rest in great measure upon the prompt recognition and proper evaluation of the four chief symptoms enumerated.

Palpating Hernial Rings and Rectum

Fortunately for positive diagnosis, about half of all obstructions of intestines occur outside the abdomen, within hernial orifices.[192] (See Chart 1.) Here careful physical examinations should reveal the seat of the trouble (Fig. 149). And since all other causes combined scarcely equal in frequency this single cause, painstaking examination of the sites at which herniae occur becomes an imperative necessity whenever bowel stoppage is in the least degree suspected. How often do cases come under observation in which a patient is suffering from pain, vomiting, constipation, or distention and in which no diagnosis has been made, yet in which hernial rings, particularly the femoral, have had no examination! The incidence of hernial strangulation or obstruction among etiological factors does not warrant neglect of this particular routine of inspecting and palpating all sites for rupture. The physical test of next importance is rectal examination. This may reveal rectal cancer; fibrous, syphilitic or tuberculous stricture; sigmoid volvulus;

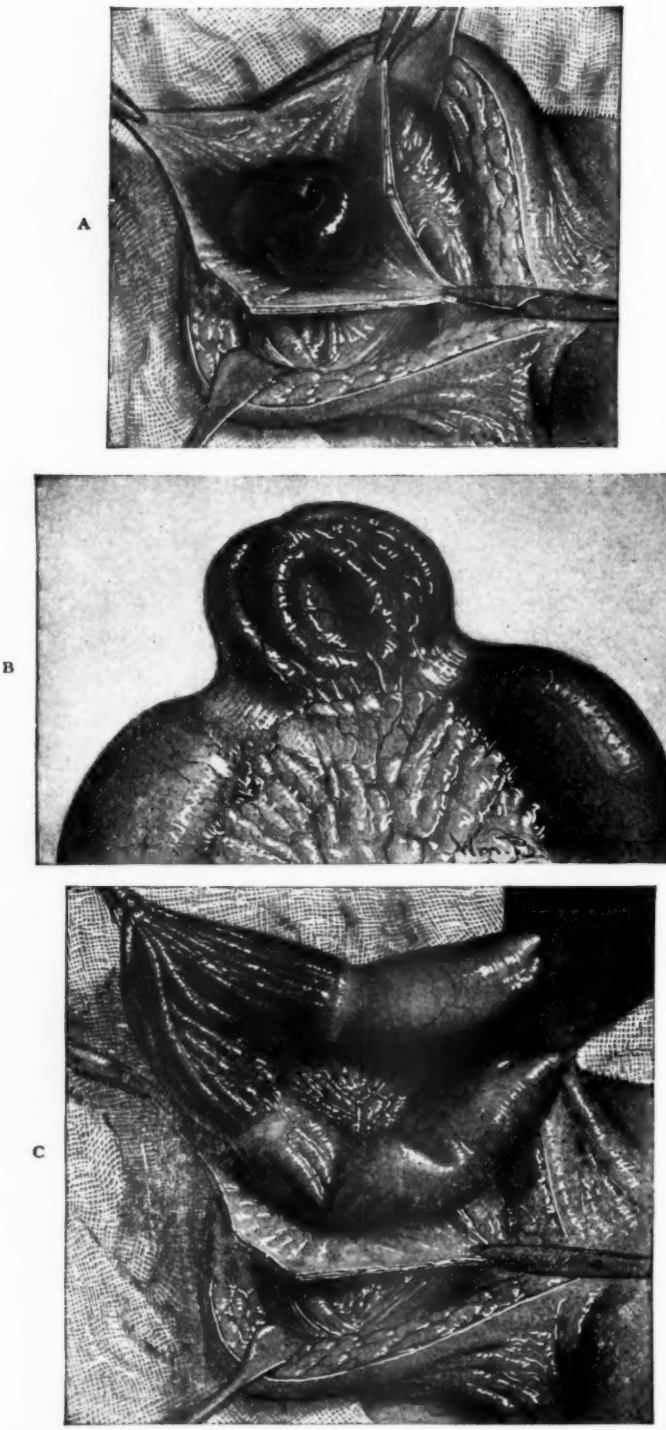


FIG. 149. (From Babcock's Text Book of Surgery, W. B. Saunders & Co.)

A. Strangulated femoral hernia. The sac has been opened, blood-stained fluid has escaped, the strangulated loop of ileum, although dark red or nearly black, is shiny, resilient, and viable.

B. Strangulated loop of bowel from hernial sac showing constriction from pressure at the hernial ring, the gut is viable. A strangulated loop should always be pulled well out of the wound to make sure that the constriction at the abdominal ring has not produced necrosis. Bowel reduced with radical herniorrhaphy without drainage.

C. A strangulated loop of bowel withdrawn from hernial sac showing grooves from pressure necrosis. Resection of intestine, enterostomy, or the formation of an artificial anus is necessary.

or colic intussusception. Since rectal cancer is the most common cause of chronic intestinal obstructions (and of acute engrafted upon chronic) and 96 per cent of rectal cancers[193] are within reach of the examining finger, it is clear that rectal digital examination combined with palpation of hernial orifices may be looked to as a means for establishing a positive diagnosis in the majority of instances of intestinal obstruction.

Additional Physical Findings

Physical examination may reveal other evidence. Tumors are sometimes felt or seen. With acute obstructions intra-abdominal tumors are found in but 16 per cent of cases although with chronic obstructions the incidence of masses rises to 54 per cent.[194] Often the mass is a malignancy, the commonest location being retrosigmoid, next cecum or ileocecal junction, then other parts of large bowel, and least often, small bowel. Diffuse tympanytic masses are caused, though rarely, by volvulus (ileojejunal or sigmoid) or occur in conjunction with an internal hernia (into lesser sac; paracecal or paraduodenal fossa). The tumor subject to easiest identification is the sausage-shaped mass of intussusception. Characteristic distentions have been mentioned (central with small bowel blockage; in flanks with large gut stoppage; local with volvulus and internal herniae; diffuse and massive with adynamic ileus). Ladder patterns and visible peristalsic waves which pass regularly to a fixed point and there abruptly terminate have been noted. Inspection may also reveal scars of old operations which suggest underlying band obstruction; while the rare observation of stigmata of congenital defects (high palatine arch; cleft palate; hare lip; polydactylism) brings to mind the possibility of a persistent omphalomesenteric duct (Meckel's diverticulum). Auscultation, it has been stated, at times reveals excessive gurgling and splashing sounds with acute mechanical obstructions and a deadness or silence with adynamic and spastic ileus. Percussion yields little information of value.[194] Rigidity is usually absent with early obstruction or may involve so small a segment of belly wall, be so moderate in degree, or prove so difficult to differentiate from the voluntary

rigidity due to pain and efforts with vomiting that its usefulness in diagnosis is not great. Sensory disturbances, also, prove of but little value since scarcely ever is an identical segment of bowel trapped or blocked in consecutive cases. And local tenderness is sometimes present and sometimes absent; in the former instances it is hard to distinguish from that due to peritonitis. Indeed peritonitis is often associated; for peritonitis may lead to intestinal obstruction (adynamic type) and obstructions (mechanical variety) lead to peritonitis. Systemic examinations usually reveal little which is typical of intestinal obstruction alone. In general, the slight value of the physical examination, or its absolute lack of any clear cut value in a considerable group of cases forms a sharp contrast to its positive worth when a strangulated hernia, a rectal neoplasm, or a sausage shaped tumor is discovered.

Etiological Classifications

The true clinician, when attempting a diagnosis, bears continually in mind a classification of causes and he is acquainted with the law of chance or the probabilities regarding these causes; he is guided by established clinical rules. The knowledge, for example, that herniae dominate other etiological factors in obstruction puts him on guard relative to this dangerous condition, for every hernia may be considered as a potential intestinal obstruction; and to know that there are among the long list of possible causes but three other *common* types, each of which is quite rigidly restricted to a certain age group, increases his accuracy in preoperative diagnosis as nothing could do but a statistical study of known operative findings.

For a study of any specific case of suspected intestinal obstruction the following classification of possible causes is serviceable (see Figs. 150 and 152):

A. Acute Obstructions

1. Mechanical Types

a. External hernia (inguinal, femoral, umbilical, incisional, etc.).

b. Adhesions or bands (previous peritonitis; sites of operations).
c. Intussusceptions (ileal, ileocolic, colic).
d. Carcinomata and other neoplasms (chiefly in rectum or sigmoid).
e. to i. Volvulus (sigmoid, jejunoileal).
Internal herniae (lesser sac; diaphragm; fossae, etc.).
Duodenomesenteric ileus (postoperative; with visceroptosis).
Meckel's diverticulum.
Obstipations (impacted gallstones; enteroliths).

2. Non-mechanical Types

a. Adynamic or paralytic (usual cause, peritonitis).
b. Dynamic or spastic (reflex from blow) (seldom surgical).
c. Combinations of spastic and paralytic (as with thrombosis of embolism of mesenteric vessels).

B. Chronic Obstructions

Newgrowths
Strictures
Adhesions
Impactions
Hirschsprung's Disease

Of mechanical types, generally speaking, herniae cause half of cases, and there are but three other *common* forms, namely, (1) intussusceptions (over 75 per cent of which occur during the first two years of life), (2), cancers (occurring usually in middle or late life) and (3) peritoneal bands and adhesions (affecting any who have had peritonitis or abdominal operations).[192] (See Chart 1.) All other varieties of mechanical obstructions combined (internal herniae, diverticula, impacted stones, volvulus, mesenteric thrombosis, etc.) account for but from 15 per cent to 20 per cent of instances.[195]

To memorize causes of obstruction upon a purely anatomical basis as (a) those affecting bowel lumen (impactions;

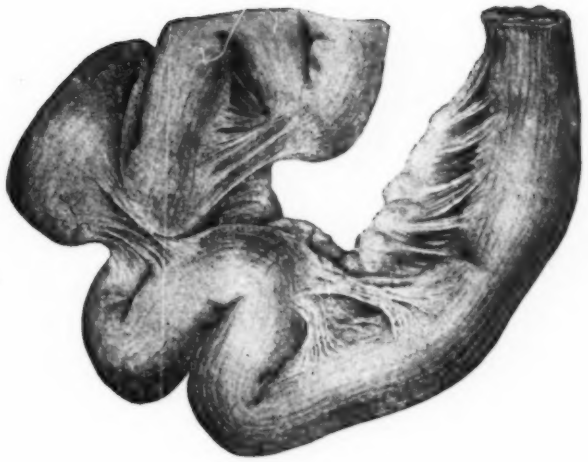
A

B

FIG. 150. Various forms of intestinal obstruction.
(A, B and C From Walton's Surgical Diagnosis, Wm. Wood & Co.)

A. Acute intussusception.
B. Acute intestinal obstruction from a gallstone.

C



D

FIG. 150.

C. Strangulation of intestine by a band.

D. View of the cicatricial adhesions and bands of the convexity and mesenteric insertion of the agglutinated intestinal coils (Payr). (From Keen's Textbook of Surgery, W. B. Saunders & Co.)

E

F

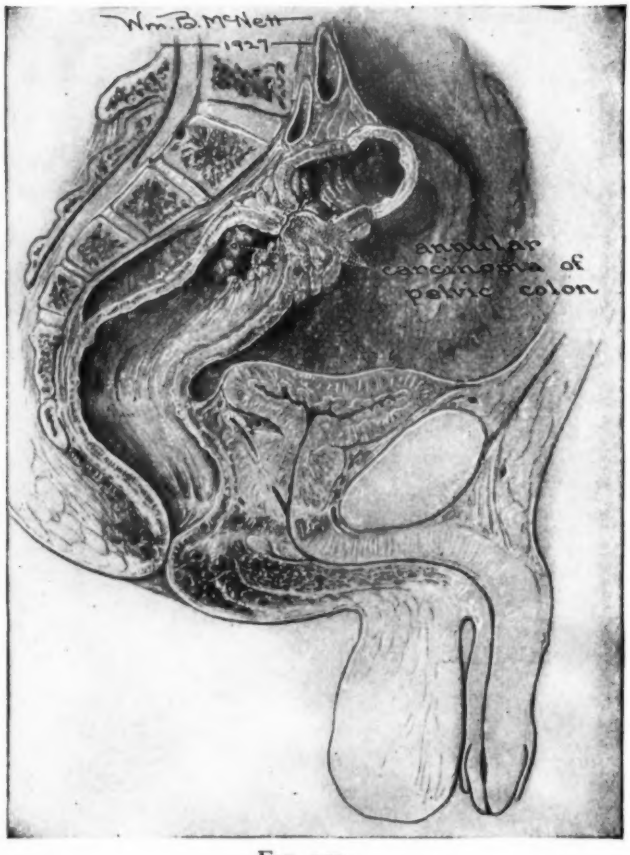


FIG. 150.

E. Hernia into the fossa duodeno-jejunalis (Treitz). The hernial orifice is displaced to near the cecum. The colon has been drawn aside to show the sac which contains all the small intestine. (From Treves' Manual of Operative Surgery, Lea & Febiger.)

F. Annular scirrhous carcinoma of the pelvic colon producing intestinal obstruction. (From Babcock's Textbook of Surgery, W. B. Saunders Co.)

polypoid or benign tumors, etc.) (b) those affecting bowel walls (cancers, strictures, Hirschsprung's disease, etc.) and (c) those affecting the intestine from outside (adhesions, bands, tumors, etc.) is of relatively little use for the clinician who must associate with a given cause an idea as to its likelihood of occurrence. Clinical rules like all generalizations serve as but rough guides and are not free from flaws; neither does it matter with the particular patient who actually has one of the more rare forms of obstruction that the majority of other patients have some other single cause; and general rules are by no means substitutes for painstaking historical and physical examinations; but the proved laws of chance are invaluable in indicating the likely clinical diagnosis, and they ensure that the trained clinician approach the individual case in a manner which is systematic with regard to what previous experience with intestinal obstruction has taught. Restating, then, and regrouping in various ways clinical rules concerning acute intestinal obstruction:

Clinical Rules and the Laws of Chance

With all patients except infants the possibility of a hernial obstruction or strangulation should be the first thought.

The most common surgical emergency in infancy (under two years) is intussusception.

Given an infant with evidences of intestinal obstruction, the likely causes, in order of frequency, are intussusception, hernial obstruction, and blockage from some congenital defect such as a persistent Meckel's diverticulum; but of all causes intussusception greatly predominates.

The most commonly overlooked physical evidence of an intestinal obstruction is an incarceration or strangulation of a femoral hernia.

A history of a previous attack of appendicits, salpingitis, cholecystitis, or some other peritoneal inflammation gives a suggestive lead as to the site of a subsequent intestinal obstruction.

A history of preceding common duct obstruction brings to mind the possibility of obturation from a gallstone; a history of

syphilis suggests a gummatous infiltration or stricture; a history of tuberculosis indicates the chance of a tuberculous enteritis with ulceration or stricture formation or of a tabes mesenterica with pressure on the bowel from without.

With a visceroptotic individual volvulus is more likely than with the normal.

Evidences of heart disease or aortitis or of embolic phenomena elsewhere point toward plugging of the superior mesenteric artery.

Obstruction occurring in the presence of acute peritonitis is most likely to be adynamic in type.

With the aged after herniae, malignancies of the large bowel are to be thought of and a rectal examination is always indicated.

Although the operative mortality with strangulated herniae is lower than that with most forms of operations for acute obstruction strangulated hernia still is the most common cause of death with acute obstruction.

High intestinal obstruction occurring immediately after a laparotomy is likely due to duodenomesenteric ileus; obstructions occurring in those who have undergone intra-abdominal operations are often caused by peritoneal band or adhesions.

When on physical examination no hernial, rectal, or abdominal mass is found, and when the patient has had neither peritonitis nor an intraperitoneal operation one of the rarer forms of obstruction is to be considered.

With definite evidence of acute intestinal obstruction when a diagnosis of the cause cannot be established from a careful historical and physical examination, immediate operation to attempt the diagnosis by intra-abdominal exploration is definitely indicated, for treatment must be prompt if the life is to be saved.

Summary

To summarize and to repeat for emphasis salient points regarding the clinical diagnosis of intestinal obstruction:

Two questions must be answered; (1) has an obstruction occurred? and (2) what is the probable nature and site of the

obstruction? To answer the first question chief reliance is placed upon history. If pain, vomiting, constipation, or abdominal distention are complained of, by cross examination seek to settle whether the pain is colicky, the vomiting regurgitant, the constipation absolute, or the distention steadily increasing (qualifying points of the utmost importance). Ascertain the exact site or sites to which pain is referred. Give special attention to previous operations or attacks of peritonitis (appendicitis; salpingitis, etc.); also to a history of hernia, syphilis, tuberculosis, marked loss of weight, common duct obstruction; study with great care an account of the expulsive activities of the alimentary tract (increasing constipation, constipation alternating with diarrhea, blood, mucus, tenesmus, ribboning of stools). Examine all hernial orifices and the rectum. Search carefully for intra-abdominal masses. Recall that a totally negative local or systemic examination does not necessarily rule out obstruction. When the history or the examination points to the presence of a bowel stoppage keep in mind the laws of probability in reaching a clinical opinion as to the likely site.

Early Diagnosis and Prompt Operation

Refinements as to diagnosis need not be carried too far. Early diagnosis as to the blockage itself is the most important matter. Time spent in prolonged laboratory tests with this unusually serious condition, or in extended efforts by means of repeated enemata to prove the presence of absolute constipation, is frequently time taken at the expense of the patient's life expectancy. With acute obstructions treatment by operations must be prompt for each hour adds to the risk. Perhaps in no other surgical emergency is procrastination more disastrous and the necessity for a serious attempt at an immediate diagnosis upon the basis of information at hand at the first examination more pressing. There are but few instances of acute intestinal obstruction in which the underlying condition can not be relieved or the condition of the patient alleviated if operation is undertaken within the first twenty-four hours of onset of the acute symptoms.[196]

Mortality with Obstruction

The mortality with acute intestinal obstruction is out of all proportion to that from any other common surgical affection of the gastrointestinal tract. Until recently, considering all causes of obstruction together, with the most approved methods of treatment more patients died than recovered (an immediate mortality of over 50 per cent). And even a few most recent statistical studies show a mortality rate after operations, grouping all types of obstructions with the exception of hernial strangulations, which ranges from fifty to 80 per cent[197] and without prompt relief bowel stoppage, once complete, tends to be uniformly fatal. Spontaneous recovery from mechanical blockage is so rare an occurrence that it can scarcely be looked for. There is one type of obstruction which responds with almost certainty to non-surgical treatment, namely duodenomesenteric ileus (see page 218); and intussusception has been dealt with in some instances by means of external manipulations.[198] But with these exceptions there is fair agreement that operative intervention represents the only reasonable chance for recovery. By comparison with results now obtained from operations for other serious abdominal diseases the mortality rate with obstruction still is appalling and constitutes a challange to concentrated surgical research and study. It has been stressed that the chief factor in obtaining improved results is early diagnosis followed by prompt operation. And as this doctrine has gained in acceptance the general mortality rate has been correspondingly lowered. The most encouraging statistics available record an immediate death rate of but 16 per cent when operations were performed within twenty-four hours of onset of acute symptoms, (and among cases in this list were several with thrombosis of mesenteric vessels) and of but 30 per cent with operations performed at varying periods after onset.[199] An appended chart reviewing over 3000 cases demonstrates a mortality rate, which may still be considered as unusually low, of less than 20 per cent for hernial obstructions and 35 per cent for remaining grouped cases, (see Chart 1).

A. J. S., n. s. Vol. IX, No. 3, p. 618

CHART 1

ANALYSIS OF 3064 CASES OF INTESTINAL OBSTRUCTION (SOUTTAR)

Cause	Site				Operation					Result	
	Small Int.	Right Colon	Left Colon	Rect.	Reduction	Enterotomy	Enterostomy	Anastomosis	Resection	R.	D.
Gall stones	26	...	2	..	...	25	3	..	..	14	14
Carcinoma	2	46	246	54	...	..	313	48	37	202	156
Adhesions	308	23	10	2	283	..	..	36	20	235	107
Internal strangulation	211	6	6	..	134	..	27	32	33	149	74
Intussusception (idiopathic)	526	100	14	..	544	2	13	48	58	476	137
Intussusception with tumor	39	18	20	..	47	3	17	12	18	36	38
Total (excluding hernias)	1655										
Omental											
Inguinal hernia	457	31	13	29	501	2	11	27	34	451	83
Femoral hernia	537	5	4	29	618	1	14	55	57	557	118
Umbilical hernia	159	36		12	170	1	14	27	33	129	71
Total hernias	1409										
Grand total	3064										

Intraperitoneal Exploration

A routine for intra-abdominal exploration might be summarized thus; open the peritoneal cavity near the site of a hernia, old operative scar, local mass, or other quite certain site; otherwise employ a right midrectus incision which may later be elongated in either direction. Before handling anything gain all possible information by inspection (look for gangrenous or discolored gut, for greatly enlarged loops, for stringy bowel). Search for the site of obstruction in one of three ways; (a) allow a loop of intestine to present into the wound; note which end presents most easily and quickly and which slowly and with more difficulty, (the latter probably leads toward the obstruction); (b) if stringy or collapsed bowel is observed follow it orad to find the site of constriction (see p. 350); (c) if greatly dilated and discolored loops are found the site of obstruction is probably where the distention and discoloration are greatest. Examine all hernial rings, both external and internal (inguinal, femoral, umbilical, foramen of Winslow, fossa of Treitz, paracecal and parasigmoid regions; diaphragm,

etc.). Slip the examining hand to the left lower quadrant, especially where all loops both of small and large bowel seem enlarged and examine the iliac colon, pelvic colon and upper rectum. See that all loops of small bowel are free from the parietes and may be displaced en masse to the right and left on the mesenteric hinge. Trace the root of the mesentery, seeking mesenteric slits. Follow systematically foot after foot of bowel if need be, particularly the last 5 to 10 ft. of ileum if Meckel's diverticulum is to be excluded. When necessary, incise and drain distended bowel (see p. 309); after repairing the bowel opening, re-examine the peritoneal cavity for additional information made available by the collapsed condition of the intestine. Be persistent, find the cause or rule out an obstruction even with poor risk cases (overlooked gangrenous loops or tight constrictions spell quite certain death). With acute mechanical obstructions, only in desperate cases does it seem justifiable to blindly make a temporary stoma in a presenting loop and withdraw. Exercise the utmost gentleness, even when explorations are extensive, avoiding evisceration and needless handling of gut. Show respect for already damaged tissues.

Experimental Data

Experimental studies regarding acute obstruction have been unusually extensive and they hold promise of leading in the near future to materially improved end-results in clinical cases, for already methods have been devised for definitely prolonging the lives of the laboratory animals. But so technical are the problems involved and so conflicting the data, that adequately to review such work falls distinctly beyond the scope of this book. To make brief additional references, however, to the chief causes of death with acute obstructions and strangulations (a, the element of toxemia; b, the factors of dehydration and disturbance of the acid-base equilibrium) serves the dual purpose of bringing out further points regarding gastrointestinal physiology and of emphasizing essential practical details in treatment. Appended bibliography also indicates authoritative sources from which to obtain information relative to this subject.[200]

A. J. S., n. s. Vol. IX, No. 3, 620.

Older Theories and New Facts

Death from intestinal obstruction originally was attributed to shock, extreme loss of fluids from the body and starvation. The fluid loss and the inability of replacement through natural channels are so obvious and impressive a factor with acute obstructions that these were long taken for granted as outstanding in determining the fatal issue. Deaths which occurred so promptly that vomiting had not begun were explained upon the basis of shock or nerve exhaustion from pain and the overwhelming of vital centers by a rapid and steady flow of afferent stimuli. Suspicion that these explanations were inadequate to account for all the facts was aroused by the demonstration that simple replacement of lost fluids with water fails to prolong life; and that treatment with water and alkalies, the latter to combat a supposed acidosis as judged to be present by the dry tongue and the typical odor to the breath after prolonged vomiting, definitely hastened, rather than retarded death. There tends to be present, it was shown, an alkalosis rather than an acidosis, the odor of the breath being caused by the presence of acetone and other ketotic acids (a ketosis) due to the incomplete combustion of fats with the starvation which followed the constant vomiting. Thus there is found present a high rather than a low CO_2 combining power of the blood; and whereas glucose seems indicated, sodium bicarbonate and other alkalies are definitely contraindicated. Chemical tests of blood show, furthermore, a marked decrease in the chlorides; also a sharp rise in the non-protein nitrogen content, chiefly in the form of urea. The chloride decrease appears to be uniformly present with simple bowel obstruction and the surprising fact seems demonstrated that it does occur where there is no vomiting. With certain of the experimental strangulations of the bowel no loss of chlorides occurs. The rise in non-protein nitrogen, upon the other hand, is present both with strangulations and with simple obstructions, but with the latter it occurs only *after* the fall in blood chlorides while prevention of the chloride fall prevents nitrogenous changes and prolongs, or even saves the life of the animal. Retention by the kidneys

of the nitrogenous compounds, it has been shown, does not account for the increase in the blood and apparently there is an actual new creation of these end-products of protein decomposition in excessive amounts at some point in the body. Suspicion that with intestinal obstructions, especially when high in the tract, some specific toxin is present was aroused by the observation that when normal animals were injected with material taken from strangulated or obstructed loops of other animals they promptly died after exhibiting almost all evidences of having had intestinal obstruction. Other experiments have demonstrated that such toxic material is not absorbed by normal mucous membrane, for several hundred times a lethal dose may exist within bowel lumen, yet the animal remain well provided the mucosa is intact. Absorption quickly takes place, however, when the membrane is injured; and once the lethal dose has entered the circulation no treatment of the obstruction itself nor any known form of supportive or other treatment is capable of preventing death. The nature of the specific toxin (denied by some who believe that the intestinal contents even when taken from non-obstructed parts of the duodenum or jejunum may be shown to be toxic) remains unknown notwithstanding much study regarding it. As to the source of the toxic material the following have been suggested; the bowel mucosa, the pancreatic juice, intestinal bacteria, etc.; and as judged by its actions when injected into normal animals it has been considered a toxic proteose, a histamine, activated pancreatic fluid, a bacterial toxin, etc. Experimentation has clearly demonstrated that symptoms begin earlier and death occurs more promptly when the point of obstruction in the bowel is located immediately below the opening of the common bile duct and pancreatic duct; also that the severity of the condition bears an inverse relationship to the proximity of the point of obstruction to this greatest danger zone. And bowel strangulations, as might be expected, have been shown to yield a far more acute clinical picture and to cause much more rapid death than do simple obstructions

to intestinal lumen without concomitant damage to the circulation.

Practical Suggestions

This tremendous amount of investigation and the many theories regarding the toxic element with obstruction have not yet resulted in the discovery of any practical means for preventing death in clinical cases. At present there are available no confirmed statistics which demonstrate convincingly the value in reducing mortality of any method of treatment for acute and complete mechanical obstructions other than early surgical intervention. Limited figures with reference to the use of B. Welchii antitoxin are encouraging.[201] And the use of sodium chloride seems, upon experimental grounds and from early clinical enthusiasm, to be gaining a position as an established addition to therapeutic measures;[202] yet too few observations concerning its clinical use are available to form the basis for fair conclusions regarding its effect upon the death rate. It has been proposed that tests of the blood chlorides afford an index to the degree of the toxemia present for certain experiments suggest that the depletion of chlorides precedes protein destruction with the subsequent rise in the non-protein elements in the blood. The wisdom of repeated gastric lavage appears firmly established both upon experimental and clinical grounds; not only are toxic substances thus removed but in addition intraluminary pressure is relieved and further damage to mucosa presumably diminished or prevented, which is an important matter in view of the knowledge that excessive pressure within the bowel, as is that without, is capable of damaging the capillary blood supply to the lining membrane of the bowel with a secondary increase in the absorption of toxic products contained within.[203] Gentleness in handling obstructed loops seems also essential in view of the fact that toxic absorption only takes place after the bowel walls have been damaged. The general results from the use of stomata connecting bowel wall with the body surface have been disappointing and it has been pointed out that bowel motility is needed to force the contents to the stoma after one has been created,

Sodium Chloride with Obstructions

whereas with most cases of obstruction motility has either been lost or greatly impaired by the time of operation.

Sudden Toxic Absorption

At times death occurs suddenly during the course of an operation for the relief of a strangulation and the frequency of this accident immediately after the bowel has been liberated indicates the possibility that there has followed as a result of the liberation of gut, a sudden absorption into the circulation of a lethal dose of the highly toxic contents of the previously strangulated loop. Accordingly it might be advantageous before the loop is liberated, especially when the case is one of long standing, first to aspirate the loop contents.

Concerning the treatment of acute intestinal obstruction the following generalization might be made: Since all of the physiological and chemical changes noted with the condition (vomiting, dehydration, toxemia, disturbance of acid-base balance) are secondary in nature, the first consideration, now as always is the immediate surgical relief of the bowel occlusion or strangulation itself.

Intestinal Neoplasms

Foregoing points relative to the diagnosis, management and experimental findings with acute obstruction illustrate the importance of gastrointestinal physiology to the clinician. It is upon his knowledge of physiology, also, and of the signs and symptoms of bowel obstruction that the clinician must largely rely in making a diagnosis, or suspected diagnosis, of an intestinal newgrowth when this is situated above the rectum. Only by this alertness to detect and adequately to investigate early and relatively trifling evidences of chronic or intermittent intestinal obstructions will small bowel and upper colic tumors be discovered, particularly when malignant, in time to effect a cure. These neoplasms often develop to an advanced stage before being discovered, since here, in contrast to the rectum, the bowel content is normally liquid or but semi-solid and the intestinal current so easily passes the growth that the processes of alimentation are but little interfered with.[204] Interference with function being slight, early symptoms tend also to be slight.

A. J. S., n. s. Vol. IX, No. 3, p. 624

Chronic and Intermittent Obstructions

The evidences of chronic and intermittent obstructions vary from those of acute obstruction chiefly in degree. Colicky pain, even though mild and infrequent is the chief diagnostic

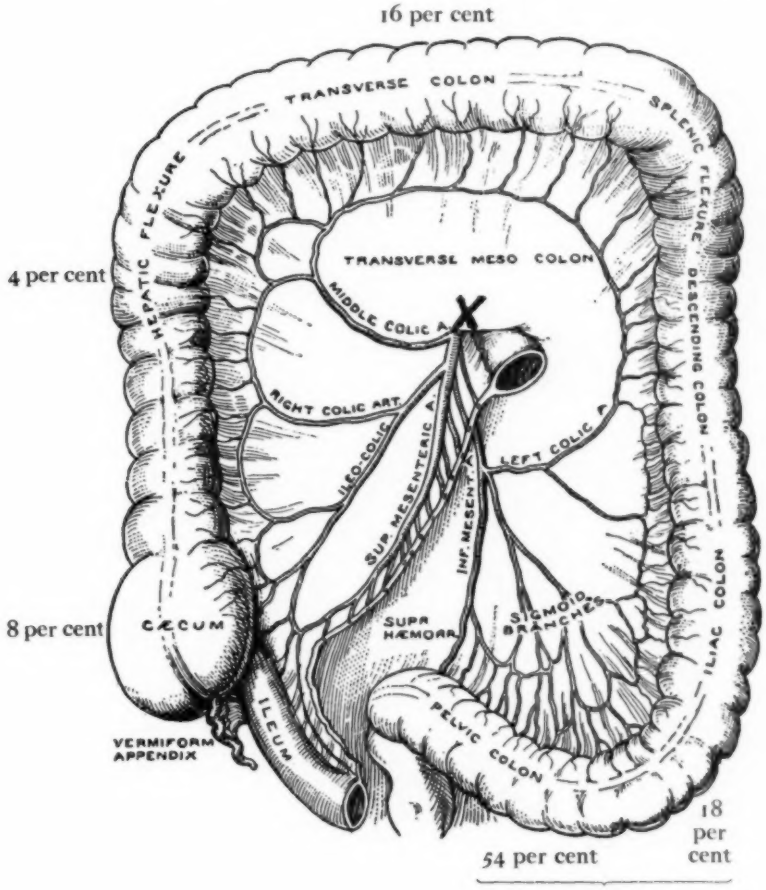
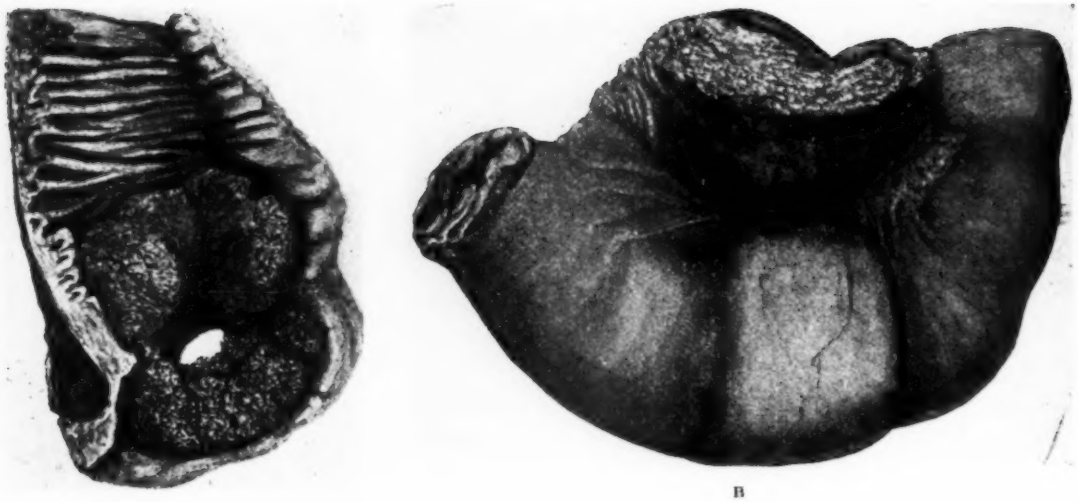
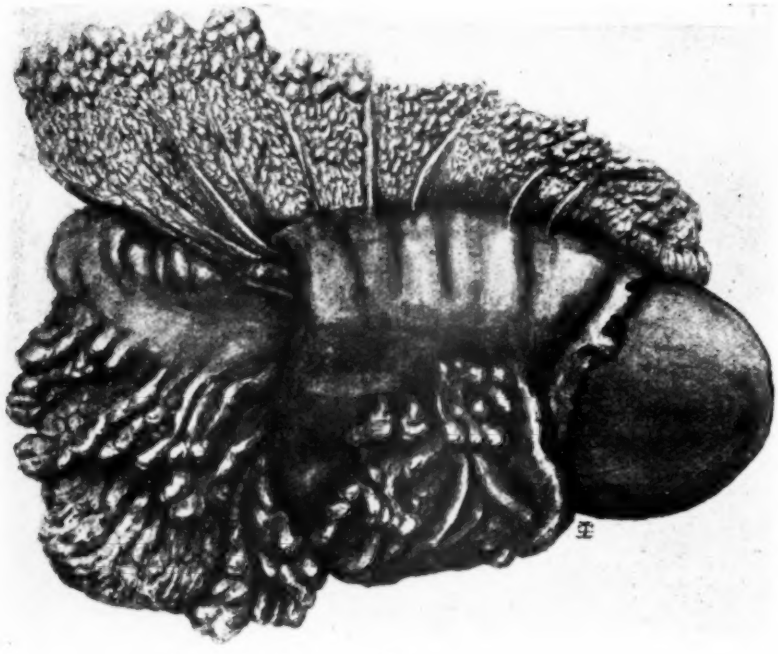


FIG. 151. Incidence of malignant neoplasms in various parts of large intestine. Figure based upon operative findings in 53 consecutive cases studied on the Third Surgical Division of Bellevue Hospital. (After Taylor's Operative Surgery.)

symptom; increasing constipation or constipation alternating with diarrhea is significant, as are also occasional attacks of vomiting. A point of great significance is the ever present anemia when the growth is malignant. This seems to be more profound the farther the growth from the rectum. And progressive anemia, slow loss of weight, a sense of vague abdominal



A B



C

FIG. 152. Enteral neoplasms. Chronic intestinal obstructions. (From Walton's Surgical Diagnosis, Wm. Wood & Co.)

A. Carcinoma of ileocecal valve.
B. Sarcoma of small intestine.
C. Chronic intussusception due to a lipoma.

discomfort (particularly when near the colic flexures or right lower quadrant) with or without evidence in the feces (occult blood, mucus, pus, cellular clusters) constitute an indication for radiographic studies, both by barium enemata and contrast meals. X-ray studies are best initiated by first giving the enema, since acute obstructions may be precipitated by the use of a heavy meal by mouth (Fig. 152).

Types of Enteral Growths

Neoplastic enteropathies may be malignant or benign. Of malignant growths carcinomata are by far the most common; less frequently encountered are sarcomata and lymphosarcomata. Of benign tumors the common forms are adenomata (polyps) and lipomata; more rare types are fibromata, myomata, angiomata, and gaseous cysts.[205] An accompanying chart illustrates the incidence of these growths at various parts of the intestinal canal (Fig. 151 and Chart 1). As will be noted, rectal and sigmoid tumors far outnumber other intestinal growths. But for the diagnosis of these, rectal digital and sigmoidoscopic examinations almost always suffice. And since these neoplasms involve peripheral nerves and lie close to the anus the diagnosis tends to be more promptly established; for among factors calling attention to these masses are perineal, sacral and lumbar pains; mucous, pus and blood by rectum; tenesmus and a sense of unfinished defecation. Such secretomotor reflexes and referred pains are by no means so common with intestinal tumors situated elsewhere.

Intestinal Masses

Unfortunately for end-results a palpable mass is already present in nearly half of the instances of high enteral growths by the time the patients seek advice. Masses at the colic flexures often prove difficult to detect and are always hard to identify since they are quite fixed and lie near viscera which are themselves not infrequently the seat of tumefactions (liver, spleen, kidney). And with neoplastic masses in the right lower quadrant differentiations from periappendiceal inflammatory masses or ileocecal tuberculosis may prove impossible by physical signs alone. Small bowel tumors usually display great mobility in all directions in contrast to relatively fixed

pancreatic growths; to biliary and cholecystic tumor which if movable swing upon an arc centered in the right upper quadrant; to splenic, nephritic and pelvic growths with which

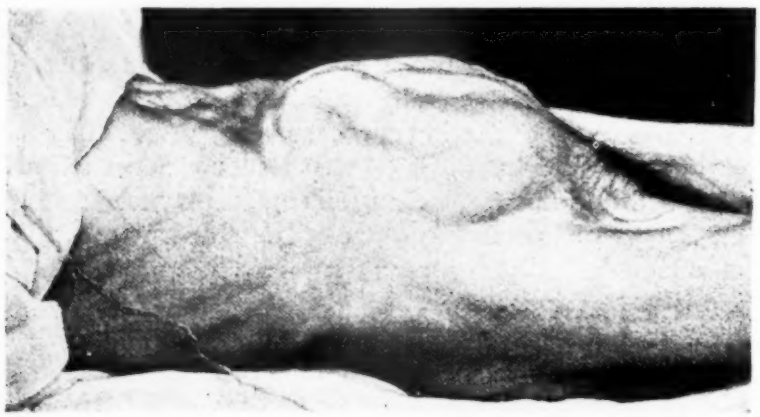


FIG. 153. Rigid contraction of bowel from obstruction of small intestine by a fibrosarcoma. (From Quervain's Surgical Diagnosis.)

characteristic limitations of motion suggest respective points of origin; and with mesenteric growths which move most freely from side to side upon the mesenteric hinge but little or not at all in the upward and downward direction. Small bowel tumors are as the rule not larger than a hen's egg although at times, in spite of the normal fluidity of bowel contents fecal accumulations occur proximal to the growths (Fig. 153). Fecal impactions without tumors are exceedingly rare except within sigmoid colon or rectum, and the presence of a mass having the size and movability described, constitutes presumptive evidence of the presence of a newgrowth. While intestinal tumors are usually primary, the type of tissue making up the mass cannot be determined by local examination and even proves difficult on gross examination of the mass itself. It is to be borne in mind that malignancies of intestine occur at relatively early periods of life, nearly one-third occurring before the age of forty. And it bears repetition that because upper alimentary neoplasms remain so long clinically quiescent on account of the fluidity of the contents of the secretory and absorptive functional divisions of the tract, radiographic

studies should be promptly made whenever vague gastrointestinal symptoms, particularly those suggesting mild or chronic obstructions, are complained of. Early detection is all the more vital since neoplasms located within the small bowel are from the technical standpoint, most advantageously situated for complete removal.

Applied Physiology

The aim of the present chapter on physiological divisions of the gastrointestinal tract has been to stress in broad terms functional activities at various parts of the alimentary canal. With reference to operative surgery practical points were discussed regarding the creation and management of fistulae, stomata, and short circuits; stressing the necessity for keeping alimentation as nearly normal as consistent with pathological conditions. And concerning surgical diagnosis, functional activities were reviewed which serve to clarify common signs and symptoms of intra-abdominal disorders.

It is taken for granted that the surgeon would be less interested in a systematic resume of physiology as such (i.e. first to bowel motility; then of secretions; then of biliary and pancreatic functions, etc.). This he can readily find authoritatively given in physiological textbooks. But such resumes contain little or no references to surgical applications; and the problem for the clinician is one of correlation. They do contain, however, much that is controversial; and of controversies the surgeon finds quite enough within his own domain. The busy practitioner, whether right or wrong, is frequently impatient with needless details and with pure pedantry; he is even willing to remain somewhat uninformed as to theories; he seeks first and sometimes only, ideas which serve adequately to explain his problems, and aids which have been proved actually to *work*, for the benefit of his patients. In a word, the clinician evaluates physiology in terms of applicability.

Applied physiology, then, remains a keynote throughout succeeding pages. And the present introductory chapter, pointing out certain physiological aspects of surgical gastroenterology, indicates the method which will subsequently be

employed in dealing with topics to follow. The discussion of functional divisions of the alimentary tract, as here given, is to be considered incomplete and unfinished. For under the heading of vascular and lymphatic absorption, next to be reviewed, will be taken up the physiology of nutrition (enzymes, digestion, selective absorption of mucous membranes); and in the chapter on visceral neurology will be found the subjects of gastrointestinal motility and reflex actions (peristalsis; peristaltic rush; ileogastric reflex); while the physiology of the portal circulation and of bile production will be dealt with in a section devoted to a study of the liver; and similarly pancreatic physiology has been assigned separate space. In short, the discussion of functional processes within the gastrointestinal tract and its derivatives is so distributed as to ensure space in which adequately to deal with practical clinical applications.

QUESTIONNAIRE

1. What are the great functional divisions of the gastrointestinal tract? Give boundaries.
2. What are the three chief phases of alimentation?
3. What is meant by foregut derivatives; midgut derivatives; hindgut derivatives?
4. Name the point in the gastrointestinal tract which roughly separates the functiona foregut from the functional midgut.
5. What is meant by the absorptive division of the alimentary tract? What division lies cephalad? What caudad? Roughly identify these divisions anatomically.
6. Why cannot the duodenum or transverse colon be considered as functional units?
7. What is the arterial blood supply of the various functional divisions of the gastrointestinal tract? Describe their nerve supplies.
8. To what degree do functional bowel divisions overlap?
9. What enzymes or digestive ferments are found in the gastric juice?
10. What substances are absorbed by the gastric mucosa?
11. What substances are absorbed by the mucous membrane of the rectum and pelvic colon?
12. To what extent does secretion occur within colon?
13. In the presence of bowel stasis how may the patient be nourished?
14. When was intravenous administration of fluids first employed?
15. How much small bowel may be resected without causing certain death?
16. What is the character of the discharge from a high jejunostomy?
17. What distressing signs and symptoms accompany high jejunostomies? How may these be controlled or alleviated?
18. How long should the surgeon wait for spontaneous closure of a high jejunostomy?
19. What are the digestive ferments or enzymes in the pancreatic juice? In the intestinal secretions?

A. J. S., n. s. Vol. IX, No. 3, p. 630

20. Describe several ways for dealing with surface excoriations and tissue digestion as observed with high fistulae.
21. How may the surgeon tell whether a fistula which he sees for the first time is high or low in the gastrointestinal tract?
22. What foods should be avoided by patients with intestinal fistulae? Describe a satisfactory diet for a patient having an ileostomy.
23. What percentage of feces consists of undigested food and food residues?
24. What is a mucous fistula? A Thiery-Vella fistula? An appendiceal mucous fistula?
25. Is it safe to return a closed loop of bowel to the peritoneal cavity?
26. What is an intestinal short circuit? An occlusion of bowel? An exclusion of bowel? What is meant by an excluded segment?
27. What are the indications for performing short circuiting operations?
28. What are the indications for creating temporary intestinal stomata or fistulae? For permanent stomata?
29. What is an artificial anus? At what points in the bowel may artificial ani be created?
30. Why is it of advantage to create a preliminary or temporary stoma in the presence of an acute colic obstruction?
31. What are some of the precautions necessary in performing a colic resection?
32. What factors tend to prevent spontaneous closure of temporary fistulae?
33. Name some factors in the creation of temporary stomata which favor their early closure.
34. What are the three most common surgical diseases of bowel between duodenum and rectum?
35. Give a clinical classification of acute intestinal obstructions.
36. Classify intestinal obstruction according to etiology.
37. What are the chief causes of death with acute intestinal obstruction?
38. Why is a high intestinal obstruction more rapidly fatal than a low obstruction?
39. Why is the course usually more severe the closer the bowel obstruction to the ampulla of Vater?
40. Name some of the more common causes of "high" intestinal obstruction.
41. What is meant by regurgitant vomiting? Describe the condition.
42. Describe the vomiting with high intestinal obstruction; with low obstruction. Explain the differences.
43. How much fluid passes into the intestinal lumen through the mucous membrane within twenty-four hours? What becomes of this fluid?
44. What is fecal vomiting? Its frequency? Its significance?
45. Describe various types of pain caused by intestinal obstruction. Explain the differences.
46. What is an intestinal colic? Its cause?
47. What are some of the most common causes of umbilical pain? Of hypogastric pain?
48. What is meant by a metameric or segmental distribution of pain or of cutaneous tenderness?
49. What are the chief factors causing changes in the amount and character of pain with intestinal obstruction?
50. What are the chief signs and symptoms of acute intestinal obstruction? Name the four which are outstanding. Explain each.
51. What is meant by absolute constipation? How determined?
52. What percentage of acute intestinal obstructions are due to herniae?
53. What are the four most common types of acute intestinal obstructions? The approximate incidence of each?

54. What are the two most important tests in the physical examination for intestinal obstruction?
55. What is the most commonly overlooked physical evidence of acute intestinal obstructions?
56. What percentage of rectal neoplasms are palpable by rectum? What other causes of intestinal obstruction may be found by rectal digital examination?
57. Name some of the causes of tumefactions or tumors sometimes found upon physical examination for intestinal obstruction.
58. In approximately what percentage of cases of acute obstructions are tumors or palpable masses felt? How often in chronic obstructions?
59. What are the most common physical findings with intestinal obstructions?
60. Define and describe dynamic ileus; adynamic ileus; intussusception; volvulus; obstipation; duodenomesenteric ileus.
61. What is the most common surgical emergency in the first two years of life?
62. What are the most common causes of intestinal obstruction with the aged? In infancy? In middle life?
63. What type of intestinal obstruction causes most deaths? What types have the highest mortality rate?
64. What is the general mortality rate with acute intestinal obstructions?
65. What are some of the more important factors influencing the mortality rate with intestinal obstructions?
66. Describe the intra-abdominal examination with a case of acute intestinal obstruction of unknown origin.
67. Discuss the toxemma of high intestinal obstruction.
68. Discuss disturbances of the acid-base equilibrium observed with intestinal obstructions.
69. Discuss the administration of the following in the treatment of acute intestinal obstructions; glucose, sodium chloride, sodium bicarbonate, water, B. Welch antitoxin.
70. What are the most characteristic chemical alterations in the blood with acute intestinal obstructions? Causes?
71. Discuss the advisability of gastric lavage with bowel obstruction.
72. Explain the sudden occurrence of death immediately following the liberation of a gangrenous segment of bowel.
73. What are the most common types of malignancies of intestine? The benign tumors?
74. Why are neoplasms of the right colon more difficult to detect than those situated upon the left side?
75. Name the evidences of chronic and of intermittent intestinal obstructions.
76. Give the relative percentages of incidence of newgrowths in various parts of the intestinal canal.
77. Name some common signs and symptoms of cancer of the rectum.
78. Name some common signs and symptoms of small bowel newgrowths.
79. How frequently are intestinal neoplasms palpable, (a) in the rectum and (b) in the small intestine?
80. Describe some of the characteristics (upon physical examination) of small bowel growths. Name other tumors with which they are most commonly confused. Give differential points.
81. Describe the more important laboratory tests in establishing a diagnosis of a malignancy of the intestinal tract.

A. J. S., n. s. Vol. IX, No. 3, p. 632

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SUBJECT INDEX TO VOLUME IX

(B.B.) = Bookshelf Browsing. (Bi.B.) = B iographical Brevities. (B.R.) = Book Review. (E.) = Editorial.

Acquired contractures of hand, 413
Acute hemorrhage from corpus luteum and Graafian follicle, 538
Adhesions of small intestine, roentgen evidence of, 243
Adjustable extension for fractures, 527
Anesthesia, avertin basal, 73
  in obstetrics, 76
  foundation needed (E.), 142
  general, new methods (E.), 145
  rectal, with tribromethylalcohol, 70
  spinal: fatalities, 234
  superficial cervical plexus block, for thyroidectomy, 68
Angina pectoris, paravertebral alcohol block for the relief of pain in, 88
  treatment by paravertebral alcohol injection or operation based on the newer concepts of cardiac innervation, 98
Appendicitis, acute, statistical review of 97 cases of, 512
Avertin anesthesia in obstetrics, 76
  basal anesthesia, 73
  effects of, on liver function, 82

Bell's palsy, (Bi.B.), 567
Biochemistry, and physiology, in modern medicine (B.R.), 151
Bladder and kidneys, gonorrheal infections of, 492
Breast, route of metastasis in cancer of, 478

Calculus of upper urinary tract, 484
Cancer of breast, route of metastasis in, 478
Carcinoid tumors of small intestines, 472
Cholecystitis, 126
Chronic inflammatory vesical neck obstruction, 530
Clinical radiology of digestive tube (B.R.), 363
  study of the abdominal cavity and peritoneum, 156, 365, 581
Collected papers of the Mayo Clinic and the Mayo Foundation (B.R.), 152
Colpotomy: posterior, for pelvic infection, 494
Complete dislocation of knee, 140
Compression fractures of spine, 424
Creed of a biologist (B.R.), 151

Dedication of the Montgomery Ward Memorial Building (B.R.), 153
Deformities and obstruction of duodenum, 430
Dental shadows, 552
Diagnosis, physical (B.R.), 628
Diagnostic novocaine block of sensory and sympathetic nerves, 264
Diathermy, tonsillectomy by, 294
Disease, toxic thyroid, place of radiotherapy in treatment of, 123
Dislocation, complete, of knee, 140
  of vertebrae, 560
Duodenum, deformities and obstruction of, 430

Effects of avertin on liver function, 82
Electrosurgery, use of, in neurological surgery, 207
Episiotomy as means of preserving pelvic floor during labor, 520
Eustachian tube (Bi.B.), 347
Evaluation of dental roentgenographic shadows, 552
  of *x*-ray examination in duodenal ulcer, 465

Foreign bodies in urethra, 335
Fractures, adjustable extension for, 527
  compression of spine, 424
  of knee-joint, 558
  modern hospitals (E.), 563
  pathological, in primary bone tumors of extremities, 251

Gall bladder disease, 131
Gonococcal infection in the male (B.R.), 580
Gonorrheal infections of bladder and kidneys, 492
Great painters and their works as seen by a doctor (B.R.), 152

Hand, acquired contractures of, 413
  two cases of disability of, 332
Hemorrhage, acute, from corpus luteum and Graafian follicle, 538
"Henle Tubules" (Bi.B.), 147
History, medical, of Michigan (B.R.), 579
Hospitals, modern, and fractures (E.), 563
Hyperthyroidism, non-surgical treatment of, 118
Hypertrophy, benign prostatic, medical aspects of treatment of, 502
Hypnotics, barbituric acid, intravenous use of, in surgery, 110

Indigestion, nervous (B.R.), 578
Infection, gonococcal, in the male (B.R.), 580
  gonorrheal, of bladder and kidneys, 492
  pelvic, posterior colpotomy for, 494
Injuries to joints (B.R.), 580
Innervation, cardiac, newer concepts of, treatment of angina pectoris by paravertebral alcohol injection or operation, based on, 98
Intestines, small, carcinoid tumors of, 472
Intravenous use of the barbituric acid hypnotics in surgery, 110

Joints, injuries to (B.R.), 580

Kidney(s), bladder, and, gonorrheal infections of, 492
  traumatized, 499
Knee, complete dislocation of, 140

Ligament repair, 556
Liver function, effects of avertin on, 82
Lymphangioma of mesentery, 441

Manual of physiology for students and practitioners (B.R.), 580
Mayo Clinic and Mayo Foundation, collected papers of (B.R.), 152
Medical aspects of treatment of benign prostatic hypertrophy, 502
  history of Michigan (B.R.), 579
  registration in France (B.B.), 616
  teaching (E.), 565
Medicine, modern, physiology and biochemistry in (B.R.), 151
  progressive (B.R.), 154

Mesentery, lymphangioma of, 441
Metatarsals and their injury, 135
Minor surgery (B.R.), 153

Needed—an anesthesia foundation (E.), 142
Neoplasms, prostatic, 507
Nephroptosis, 218
Nerves, sensory and sympathetic, diagnostic novocaine block of, 264
Nervous indigestion (B.R.), 578
New cystoscopic rongeur, 329
methods of producing general anesthesia (E.), 145
Nightingale, Florence, angel of mercy and human dynamo (B.B.), 148
Non-surgical treatment of hyperthyroidism, 118

Obituary: Walter M. Brickner (E.), 345
Obstetrics, avertin anesthesia in, 76
for nurses (B.R.), 153
Obstruction, chronic inflammatory vesical neck, 1
Operative repair of anterior crucial ligament of knee-joint, 556
Osteochondral fracture of knee-joint, 558
Otolaryngology, tribromethyl alcohol, 78

Paravertebral alcohol block for the relief of pain in angina pectoris, 88
Pathological fractures in primary bone tumors of extremities, 251
Physical diagnosis (B.R.), 628
Physikalische-chemische probleme in der chirurgie (B.R.), 153
Physiology and biochemistry in modern medicine (B.R.), 151
manual of (B.R.), 580
Physiotherapy in relation to surgery (E.), 338
Place of radiotherapy in treatment of toxic thyroid disease, 123
Pleistocene luxations (B.B.), 348
Posterior colpotomy for pelvic infection, 494
Progressive medicine (B.R.), 154
Prostatic neoplasms, 507
Pseudoperitonitis, 480

Radiographie nephro-cholecystique (B.R.), 362
Radiological studies of lung and mediastinum, vascular and aortic radiology (B.R.), 363
Radiologist as consultant (E.), 344
Radiology, clinical, of digestive tube (B.R.), 363
Radiotherapy, place of, in treatment of toxic thyroid disease, 123
in treatment of thyrotoxicosis, 115
Rectal anesthesia with tribromethylalcohol, 70
Renal tuberculosis, 545
Roentgen evidence of adhesions of small intestine, 243
Roentgentherapy, treatment of syringomyelia, 302
Rongeur, new cystoscopic, 329
Route of metastasis in cancer of breast, 478

Sarcoma of stomach, 444
Souvenirs of medical registration in France (B.B.), 568
Spinal anesthesia: fatalities, 234
Spine, compression fractures of, 424
Statistical review of 97 cases of acute appendicitis, 512
Stomach, sarcoma of, 444
Superficial cervical plexus block anesthesia for thyroidectomy, 68
Surgery, intravenous use of the barbituric acid hypnotics in, 110
minor (B.R.), 153
physiotherapy in relation to (E.), 338
synopsis of (B.R.), 152
Surgical diagnosis (B.R.), 152
Synopsis of surgery (B.R.), 152
Syphilis héréditaire du systéme nerveux (B.R.), 153
Syringomyelia, treatment of, by roentgentherapy, 302

Teaching, medical (E.), 565
Tendon transplantation, 331
Therapeutic nerve-block with procaine and alcohol, 278
Thyroidectomy, superficial cervical plexus block anesthesia, for, 68
Thyrotoxicosis, radiotherapy in treatment of, 115
Tonsillectomy by diathermy, 294
Transplantation, tendon, 331
Traumatized kidney, 499
Treatment of syringomyelia by roentgentherapy, 302
Tribromethyl alcohol in otolaryngology, 78
rectal anesthesia with, 70
Tuberculosis, 221
renal, 545
Tumors, carcinoid, of small intestines, 472
uterine, 289
Two cases of disability of hand, 332

Ulcer, duodenal, evaluation of *x*-ray examination in, 465
Urethra, foreign bodies in, 335
Urinary tract, upper, calculus of, 484
Use of electrosurgery in neurological surgery, 207
Uterine tumors, 289

X-ray examination, evaluation of, in duodenal ulcer, 465

AUTHOR INDEX TO VOLUME IX

Alvaraz, Walter C., 578
Andrus, E. Cowles, 502
D'Aunoy, Rigney, 444

Behan, R. J., 338
Behrend, Moses, 131
Benham, Francis Roe, 126
Boland, Frank K., 148
Bourne, Wesley, 82
Brenizer, Addison G., 430
Bruger, M., 82
Buka, Alfred J., 135
Bumpus, Hermon C., 545
Burr, C. B., 579

Cabot, Richard C., 580
Case, James T., 344
Christopher, Frederick, 424
Coley, Bradley L., 251
Collings, C. W., 484
Costolow, Wm. E., 123
Crane, Whitfield, 441

Davis, A. Hobson, 629
Davis, Loyal, 207
Delherm, L., 302
Deming, Clyde Leroy, 218
DeSouza, David, 580
Dillinger, Gregg A., 294
Doran, William G., 563
Dreyer, N. B., 82

Enfield, Charles D., 465

Falk, Henry C., 494
Ferguson, Russell S., 507
Fitch, Richard H., 110

Gillis, R. A. D., 520
Ginn, Curtiss, 512
Gould, William L., 527
Groen, B. M., 207
Guttman, Joseph R., 70

Hyams, Joseph A., 530

Jeck, Howard S., 335
Johnson, V. Earl, 538
Jones, Sir Robert, 580

Kennedy, J. W., 289
Kirwin, Thomas J., 329
Koch, Sumner L., 413
Koster, Harry, 234
Kramer, Samuel E., 530
Kretschmer, Herman L., 221
Krida, Arthur, 331, 332, 556, 558, 560

Lederer, Francis L., 78
Lilienthal, Howard, 345
Livingston, Edward M., 156, 365, 581
Lurie, William A., 552
Lyle, H. Willoughby, 580

Meland, Orville N., 123
Merrill, Theodore C., 586
Moodie, Roy L., 348
Morel-Kahn, M., 302

Pugh, Winfield Scott, 492

Reed, Charles B., 76
Richter, H. M., 115
Romberger, Floyd T., 142

Salvin, Arthur A., 478
Sharp, George S., 251
Soiland, Albert, 123
Soper, Horace W., 243
Speidel, Francis G., 73
Stevens, A. R., 484
Stout, Richard B., 68
Sutton, Don C., 118
Swetlow, G. I., 88

Tatum, Arthur L., 110
Thompson, Gershom J., 545
Thompson, J. William, 243

Waters, Ralph M., 110, 145
Weigel, Edgar W., 140
Weintrob, Morris, 234
Welton, T. S., 147, 347, 565
Wetherell, Frederick S., 480
White, James C., 98, 264
Wolbarst, A. L., 580
Woodbridge, Philip D., 278
Woodruff, Stanley R., 499

Zoeller, Adelaide, 444

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